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DIAGNOSIS AND SURGICAL TREATMENT OF CHRONIC CONSTRICTIVE PERICARDITIS

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THE surgical treatment of cardiac lesions has developed more slowly than other fields of surgery because of the difficult problems which these conditions present and the hazard associated with their operative treatment. The problems in this field of surgery are concerned not only with technical surgical considerations but also with the diagnostic difficulties encountered and it is only because of the close cooperation of the clinician and the surgeon that progress in this field has been made possible.

One of the greatest advances in the surgical treatment of cardiac lesions in recent years has been accomplished in the treatment of cardiac compression associated with chronic constrictive pericarditis. This condition was recognized in the seventeenth century and Lower[1] is given the credit for the first clinical description of the disease in 1669. For many years the treatment consisted of the conservative measures afforded other forms of cardiac disease and there was little or no response. A better understanding of the cause of cardiac failure in these cases was obtained by Delorme[2] who in 1898 first proposed a separation of the adhesions between the heart and the pericardium either by sharp or blunt dissection. This procedure, however, was not carried out until 1913 when Rehn[3] tried out Delorme's theory and operated on four patients, two of

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whom died after operation and the remaining two died within the next two years. Rehn went a step farther than Delorme had suggested and resected a portion of the pericardium as well as separating the adhesions. Sauerbruch,[4] working independently in the same year, reported a successful case of partial pericardiectomy. Brauer[5] in 1902 suggested an operative procedure which was much less formidable; it consisted of removal of the precordial bony structures so the tug of the heart would be on the soft yielding structures of the chest wall. This operation which has been termed "cardiolysis" has been done more commonly than the decortication probably because the risk was much less. The procedure is only effectual in cases in which the adhesions involve the pericardium and chest wall and the pericardium is not adherent to the heart muscle. This operation offers little if any benefit for true constricting pericardium which compresses the heart muscle. When this condition exists, the actual removal of the constricting scar from the heart muscle is the only effectual procedure.

The work of these pioneers in establishing the fact that constrictive pericarditis was amenable to surgical treatment has stimulated clinical studies and investigative work in this field. The disease now has been found to be a definite entity which is recognized as an essentially mechanical condition interfering with the function of the heart. Relief can be obtained only by surgical treatment.

Diagnosis

Chronic constrictive pericarditis is due to fibrous adhesions forming between the pericardium and epicardium; these then contract around the heart muscle and prevent the normal diastolic filling of the heart. The condition is understood readily and the basis of the symptoms as well as the symptoms and physical findings are clear if it is realized an inflow stasis of gradual increasing severity is present. The heart gradually becomes less and less able to fill adequately. Thus the onset of symptoms is insidious but progressive. Those symptoms which dominate the picture are dyspnea, ascites and edema of the legs. Dyspnea is peculiar in that it is present on exercise but absent when the patient lies flat and is by no means as pronounced as that observed in the presence of an equal degree of ascites encountered in congestive heart failure attributable to other causes. The cardiac output is reduced, the blood pressure is low and the pulse rate uniformly accelerated. The heart is normal in size or not significantly enlarged. The condition usually is not associated with valvular heart disease and hence murmurs indicative of such lesions usually are absent. Calcification of the pericardium

which was present in seven of our cases can be demonstrated by careful roentgenographic examination and practically assures the accuracy of the diagnosis. If this confirmation is lacking, the electrocardiogram may yield diagnostic information.

One of the outstanding and subjective symptoms noted in these cases is digestive disturbance. This is evidenced by anorexia, epigastric fulness and distress, and probably is due to impaired function of the liver which has been noted in all cases in our series. Finally in the advanced stage of the disease the venous pressure is elevated markedly and the circulation time is greatly increased. These last facts alone serve to distinguish the condition from cirrhosis of the liver with which it may be confused.

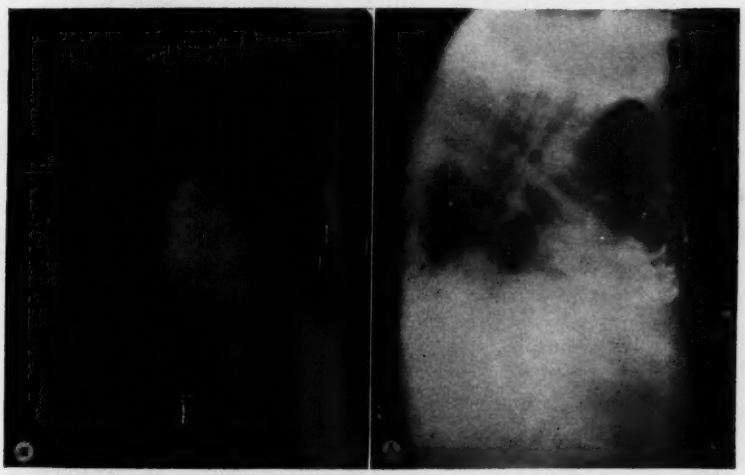

Fig. 1. Dense calcification of the pericardium on the left side anteriorly: *a*, anteroposterior view; *b*, lateral.

Our experience in the surgical treatment of chronic constrictive pericarditis consists of a series of nine cases. A brief résumé of the clinic history, operative findings and results in each of these cases follows:

Report of Cases

Case 1.—On admission to The Mayo Clinic a man, aged 38, complained of swelling of the abdomen and legs, dyspnea and palpitation. The edema had begun in the left ankle nine years previously and had appeared in the right foot two years later. Abdominal swelling had been noted five years before admission. The edema had reached its maximum in the legs five years before admission, in the abdomen two years before and since then had remained about constant. Swelling of the face after sleep had been noted for five years. Gradually increasing dyspnea had been present five years but the

patient had never experienced orthopnea. Forceful, irregular and rapid heart action on exertion had been noted for at least two years. The patient was aware that his urinary output was scanty. Digestive disturbances included constipation and gaseous distention after eating. The patient had had repeated attacks of tonsillitis, bronchitis and pleurisy and a mild attack of influenza in 1927. This illness lasted five days and was complicated by pleurisy.

Fig. 2. Sharp dissection of the calcified adherent pericardium and epicardium from the myocardium. Bone cutters were required for removal because of the dense calcification. To approach the pericardium a portion of the sternum, the cartilages and 2 cm. of four ribs of left side were removed. *Right insert,* the resected cartilage and 2 cm. of the ribs of precordial region. *Left insert,* curved precordial incision.

Examination at the Clinic disclosed a thin cyanotic man with a large abdomen and weighing 131¼ pounds (59.5 Kg.). The veins of the neck and arms were engorged. The blood pressure measured 104 mm. of mercury systolic and 74 mm. diastolic. The cardiac rhythm was interrupted by frequent premature contractions. No murmurs indicative of valvular disease were present. A pronounced umbilical hernia was observed. Marked ascites was present and the legs were moderately edematous. The teleroentgenogram did not reveal significant cardiac enlargement but disclosed marked calcification along the left and inferior borders of the cardiac shadow (fig. 1). Studies of the urine, renal function, blood chlorides, carbon dioxide combining power of the plasma, the albumin-globulin ratio of the blood and other tests on the blood yielded normal results.

On July 11, 1938, partial pericardiectomy was performed, using cyclo-

propane and oxygen anesthesia. A curved incision was made along the seventh costal cartilage to the sternum and beyond the midline of the sternum up to the third rib. The incision was extended laterally to the anterior axillary line. The musculocutaneous flap was turned back and the cartilages of the left third, fourth, fifth and sixth ribs were resected completely together with 1 inch (2.5 cm.) of each of the corresponding ribs to expose the pericardium. There was marked dilatation of the vessels over the pericardium. Some of the vessels were more than ⅜ inch (9.4 mm.) in diameter. Deposits of calcium had caused marked hardening of the pericardium.

Fig. 3. Calcified adherent pericardium removed at operation.

The pleura which was adherent to the pericardium was carefully dissected from the left portion of the pericardium lateral to the left phrenic nerve. The pericardium was extremely adherent to the myocardium and had to be dissected from the myocardium with scissors or knife. In many places the calcium was more than 1 cm. thick. It was particularly thick in the sulci between the ventricles and between the ventricles and auricles. The heart muscle was flabby, friable and yellowish in color. The left side of the heart was dissected free from the pericardium first and the left half of the pericardium was resected. The pericardium had to be cut with the rib cutters because of the thickness of the calcium which infiltrated the pericardium. The largest deposits of calcium were around the orifice of the vena cava and around the right auricle extending posteriorly along the diaphragm and in the right auriculoventricular sulcus. In the latter two situations the calcium was of two types; in some places it was granular and appeared like coarse sand; in others it consisted of firm, fixed plaques, adherent both to the diaphragmatic portion of the pericardium and to the myocardium. The larger deposits were more than 1 cm. in thickness. Such deposits had to be removed with a curet. In one pocket close to the orifice of the vena cava there was at least an ounce (30 Gm.) of this granular calcified material.

The entire right side of the heart was separated from the diaphragmatic portion of the pericardium (fig. 2). About half of the anterior part of the pericardium extending over the right side of the heart beneath the sternum was removed. Approximately half of the pericardium was removed completely and more than a third of the remaining portion of the pericardium

was dissected free from the underlying heart (fig. 3). Two Penrose drains were used to drain the pericardium. Microscopic study of the tissue removed revealed chronic pericarditis with calcification.

The patient stood the operation satisfactorily. During the entire postoperative course his temperature did not rise above 99°F. and the pulse rate did not exceed 105 beats per minute. He passed an average of 460 c.c. of urine daily on the first four days after operation, although the daily intake of fluid was as much as 2.5 liters. On the seventh day the output of urine rose and by the ninth day reached a total of 2,400 c.c. for twenty-four hours. Subsequently a negative fluid balance resulted in the rapid disappearance of the edema of the legs and of the ascites. The patient sat up on the fifteenth day after operation and was dismissed from the hospital on the thirty-third day in excellent condition (fig. 4).

Fig. 4. Patient on dismissal one month after operation. Precordial incision had healed and there was marked reduction of the abdomen owing to relief of ascites indicated by size of waistband of trousers worn by patient on admission.

The patient resumed his work as a machinist two months after operation and has remained well since. Six months after operation he returned for examination. He felt and looked well; results of physical examination were normal and he was free of edema and ascites. His venous pressure, circulation time and results of tests of liver function, all were within normal limits. This patient is essentially cured of all of his previous complaints.

CASE 2.—A man, aged 27 years, complained of dyspnea, weakness, swelling of the abdomen and edema of the legs for eight years preceding his admission to the clinic. He had had pneumonia at the age of 12 and fluid was said to have been present in the thorax although paracentesis was not performed. At the time of a second attack of pneumonia at the age of 19, a roentgenogram of the thorax revealed some deposit of lime at one border of the heart. At 21, a third attack of pneumonia occurred. Six months before the patient's admission to the Clinic a severe attack of influenza developed. Following this the edema of the ankles and the swelling of the abdomen were increased. Gastrointestinal symptoms included anorexia and postprandial distention in the epigastric region.

The physical examination disclosed a mildly cyanotic patient with a large abdomen who weighed 162 pounds (73.5 Kg.). The veins of the head and neck were distended. The pulse was irregular in rate and paradoxical in character. Its force waxed and waned with respiration. The blood pressure measured 108 mm. of mercury systolic and 80 mm. diastolic. No cardiac enlargement was demonstrated by percussion. The heart tones were satisfactory and no murmurs were present. Abdominal ascites of marked degree was observed. A sharp liver edge was felt three fingerbreadths below the right costal margin. Edema of the ankles was graded 1. Roentgen examination of the thorax did not disclose cardiac enlargement but it revealed extensive calcification of the pericardium about the apex of the heart. The concentration of hemoglobin and the blood counts were normal and the tests of renal function indicated no impairment of the kidneys. There were numerous hyaline casts in the urine.

On May 9, 1938, subtotal pericardiectomy was performed, using cyclopropane and oxygen anesthesia. A curved incision was made; the cartilages and about 1½ inches (3.7 cm.) of the third, fourth, and fifth ribs were resected. The structures overlying the mediastinum were cut through and the internal mammary artery was ligated. The pleura was thin and very adherent to the pericardium. This was separated laterally as far as the left phrenic nerve. During the separation the pleura was inadvertently opened. This opening was closed immediately with catgut. The calcified adherent pericardium then was exposed. Marked fibrosis of the pericardium which was extremely adherent to the entire myocardium was noted.

The calcium varied from a few millimeters in diameter to fully 1 cm. in some places. In some parts it was granular and laminated and was piled up in layers; between the outer and inner layers there was some semisolid granular material. Because of the thickness of the calcium over the left ventricle it was necessary to start the dissection of the pericardium to the right of the midline. After determining the true line of cleavage between the myocardium and pericardium, the dissection was carried immediately to the left ventricle.

The pericardium was completely separated from the left ventricle by sharp dissection. Then the pericardium was removed from the anterior portion of the heart. The calcium was very adherent, particularly posteriorly around the upper portion of the right ventricle and the right auricle. The right side of the heart was then completely separated from its attachment to the diaphragm and this dissection was carried posterior over the right auricle and around the orifice of the inferior vena cava. This procedure completely freed the apex of the heart. Large accumulations of granular calcium had to be removed with a curet in the course of dissection. As far as could be determined at least two-thirds of the ring of calcium around the orifice of the inferior vena cava was removed. All of the adherent pericardium was freed from the ventricles, the apex was freed completely, the right ventricle was separated from the diaphragm and the granular calcium was removed from above the right auricle and about the orifice of the inferior vena cava. In a few places the calcium had penetrated the myocardium and it did not seem advisable to remove it. Some calcium also was left attached to the diaphragm particularly around the right auricle. The pericardial cavity was drained with three small Penrose drains which were removed within forty-eight hours.

Microscopic study of the tissue removed revealed chronic pericarditis with marked calcareous degeneration.

The patient was at once placed in an oxygen tent. His immediate convalescence was satisfactory. Left pleural effusion developed which required two aspirations. His temperature did not go above 100° F. and was normal from the sixth to the twenty-sixth day, when he was about to leave the hospital. At this time his temperature rose to 101° and he complained of generalized abdominal cramps and exhibited slight jaundice. The nature of this complication could not be determined although it was thought to be due to influenza because of contact with a member of his family who had influenza. His progress following this late complication was much slower than his immediate recovery from the operation. Because his home was some distance away, he was not dismissed until two and a half months after operation.

We have not seen this patient since the operation but we have received reports in regard to his progress at frequent intervals. Almost a year had elapsed after operation before he was able to resume his activities. The last report in regard to him was received in February, 1940, and he was carrying on his usual activities. This patient is essentially cured of all of his previous complaints.

CASE 3.—A well-nourished woman, aged 31, registered at the Clinic Sept. 14, 1932, complaining of dyspnea on exertion for the past year. She had been seen at the Clinic on five occasions in the preceding four years for complaints that were minor in character and were not related to the cardiovascular system. In addition to dyspnea, epigastric distress had occurred two to three hours after eating and for the past two months she had relieved this distress by vomiting. Except for attacks of tonsillitis there was no history of previous infections.

On physical examination the veins of the neck were observed to pulsate when the patient was reclining. The liver was palpable and tender. Definite ascites could not be demonstrated. The pulse was 96 and its rhythm was regular. The blood pressure measured 110 mm. of mercury systolic and 90 mm. diastolic. No enlargement of the heart was demonstrated by percussion and no murmurs could be heard on auscultation. Teleroentgenogram of the thorax revealed extensive calcification of the pericardium. The urine contained a trace of albumin; its specific gravity was 1.030. Studies of the renal function and other tests on the blood yielded normal findings.

On April 5, 1939, partial pericardiectomy was performed on the patient, using cyclopropane, oxygen and helium anesthesia. The usual curved incision was made and the subcutaneous tissues and breast were reflected back. The costal cartilages and about 2 to 3 cm. of the third, fourth, fifth and sixth ribs were removed. The left half of the sternum was removed with a rongeur but the anterior and posterior periosteum was preserved. The left pleura was dissected carefully away from the pericardium lateral to the left phrenic nerve. It was very adherent but was separated without perforation. Then the dissection was carried down onto the pericardium which was firmly adherent to the under surface of the sternum as well as the pleura on the left side. It was difficult to find the actual line of demarcation between the pericardium and myocardium because of the firm adhesions and the extensive deposit of calcium. This deposit of calcium was between the two layers of pericardium and varied in thickness and character throughout the site of involvement; in some places it was granular and in others firm, shell-like and hard.

By careful knife dissection the true epicardium which was beyond the calcification was reached. Both layers of the pericardium with the intervening calcium were carefully dissected from the left ventricle to and including the auriculoventricular sulcus where there was a deep deposit of calcium. There was an enormous amount of calcium which was firm, shell-like, and in thin layers between the right ventricle and its attachment to the diaphragm. This was separated entirely freeing the right side and the apex of the heart. The right auricle then was separated carefully from the pericardium as far as the entrance of the inferior vena cava. A definite ring of this same deposit of calcium lined the entire diaphragmatic portion of the pericardium; this ring extended apparently around the orifice of the vena cava. A segment of this calcified ring was removed but the posterior portion of the calcium which was attached and fixed to the right auricle was not removed. The right auricle, however, was separated from the diaphragm. Approximately two-thirds of the left and a third of the right parietal pericardium and its deposit of calcium were removed and the right border of the heart was dissected from the diaphragm. The deposit of calcium and the adhesions were as extensive as they were in any case that one of us (S. W. H.) has seen and it did not seem that a satisfactory result could be obtained by a less extensive operation. There appeared to be marked myocardial degeneration. The wound was closed in layers and the pericardial cavity was drained with two Penrose drains. Microscopic study of the tissue revealed chronic pericarditis with calcareous degeneration.

The patient's postoperative course was satisfactory but prolonged. The delayed recovery was accounted for by persistent and repeated hydrothorax which required numerous aspirations. Twenty-six days after operation auricular flutter developed; this disappeared in twenty-four hours after the patient had received 10 grains (0.65 Gm.) of quinidine sulfate in divided doses. She left the hospital in good condition.

The patient was observed eight months after operation and was in excellent condition. The liver was no longer enlarged and there was no dyspnea or edema of the ankles. The heart measured 14 cm. in its greatest transverse diameter as compared to a transverse diameter of the thorax of 24 cm. The blood pressure was 118 mm. of mercury systolic and 80 mm. diastolic. The electrocardiographic tracings were normal except for an occasional premature contraction. This patient is essentially cured of all of her previous complaints.

CASE 4.—A man, aged 33, was admitted to the Clinic Sept. 13, 1938, complaining of weakness and dyspnea on exertion. His history was conspicuously negative for previous episodes of infection except for a mild attack of influenza from which he had made a prompt recovery. He had been entirely well until June 25, 1938, at which time he began to notice a sense of fulness in his abdomen and an associated soreness on pressure over the abdomen. July 20, he noted slight swelling of the ankles; by August 15, the dyspnea and weakness were becoming progressively worse, and by September 1, his physician noted definite ascites and edema of the ankles. He had lost 29 pounds (13.2 Kg.) in three months.

Examination revealed a powerfully built man weighing 195 pounds (88.5 Kg.) who did not appear ill. The pulse was regular with a rate of 72 beats per minute and the blood pressure measured 106 mm. of mercury systolic and 74 mm. diastolic. The heart was not found to be enlarged on percussion and no murmurs were present. Ascites was present and the edge of the liver

could be felt at the costal margin. There was slight pitting edema of the ankles. Extensive calcification of the pericardium was disclosed by roentgenographic studies. Pericardiectomy was advised and the patient returned for re-examination Nov. 1, 1938. The findings on physical examination were unchanged except the pulsating veins of the neck were now observed. The urine contained a moderate amount of albumin; its specific gravity was 1.026 and the concentration of urea in the blood was 30 mg. per 100 c.c. The value for hemoglobin was 10.3 Gm. per 100 c.c.; the erythrocytes numbered 4,080,000 and the leukocyte count was normal. The concentrations of blood chlorides and of serum bilirubin were normal and the van den Bergh test gave an indirect reaction.

On November 14, 1938, partial parietal pericardiectomy and segmental removal of epicardial scar were performed. Cyclopropane, oxygen and helium anesthesia was used. The usual curved incision was made with resection of the cartilages and segments of the third, fourth, fifth and sixth ribs. The left half of the lower part of the sternum was resected with a rongeur. There were extensive adhesions about the posterior surface of the sternum, mediastinal pleura and pericardium. The adhesions in this region were firm and vascular and were separated with considerable difficulty. Ligation of many bleeding points was required. The mediastinal pleura was separated laterally from the pericardium as far as the left phrenic nerve. On exposing the pericardium considerable deposits of calcium were found particularly in the upper portion of the pericardium, over the left ventricle and auricle and anteriorly beneath the sternum. They were most marked around the orifice of the vena cava, the right auricle and right auriculoventricular sulcus. The pericardium was incised anteriorly. It was difficult to find the true line of cleavage because of the calcium which had infiltrated through the fibrous scar of the pericardium and epicardium. After dissection through the pericardium a definite thin layer of scar with infiltrating calcium was found involving the epicardium. The calcium had penetrated into the heart muscle in many places. The heart muscle was injured in three places during removal of the embedded calcium and was repaired with silk. In one place there was a slight injury to the myocardial vessels which was controlled by a flap of fibrous pericardium. A considerable portion of the scar of the epicardium was removed as well as the parietal pericardium from the left ventricle and apex of the heart laterally as far as the left phrenic nerve. About a fourth of the anterior portion of the pericardium and the right side of the heart were carefully dissected from the diaphragm. On dissecting posteriorly the greater amount of calcium was found around the orifice of the vena cava and around the right ventricle. It was removed freeing the orifice. At completion of the operation about half of the parietal pericardium which was infiltrated in many places with calcium and a considerable amount of scar of the epicardium which also contained small deposits of calcium, had been removed. The operation was difficult because of adhesions and because of the firm fixed scar of the epicardium with calcium embedded into the heart muscle. The wound was closed in layers and two Penrose drains were used to drain the precordial space. The patient was given a transfusion of blood on the operating table and was placed immediately in an oxygen tent. Microscopic study of the tissue removed revealed chronic pericarditis with calcareous degeneration.

The postoperative course was complicated on the third day by an attack of paroxysmal auricular flutter associated with severe pulmonary congestion. This was controlled by the administration of digitalis and quinidine. There-

after the patient's progress was satisfactory except for a daily increase of temperature to about 101° F. associated with an increased pulse rate to 110 beats per minute. The temperature gradually returned to normal three weeks after operation and he was discharged in good condition one and a half months after pericardiectomy.

The patient was re-examined in January, 1940, fourteen months after operation. He had gained in weight and strength, experienced no dyspnea except on unusual effort and had no edema of his ankles. He was continuing to take 6 Gm. of ammonium nitrate daily and injections of mercurial diuretics at intervals of four weeks. The edge of the liver extended 3 cm. below the costal margin. The venous pressure measured 190 mm. of water and the arm-to-tongue circulation time determined by the use of decholin was 25 seconds. Obviously cardiac inflow stasis still existed but he considered that his condition was improved by 50 per cent over his preoperative state. It is possible that further operations on the pericardium will be required at a later date.

CASE 5.—A youth, aged 17, was admitted to the Clinic because of persistent ascites. He had had measles at the age of 3. At 14 he had some contact with the school prefect who had active pulmonary tuberculosis. However, several examinations of the patient's sputum had revealed nothing abnormal and it was considered that the contact had not resulted in any tuberculous infection. During his fourteenth year also the patient had an acute illness characterized by headache, malaise and an irregular febrile course. Explanation for this illness was not afforded by clinical and roentgenographic examinations. In spite of rapid improvement he remained in the hospital one month. He was in good health for the next three months. Four months after his acute illness pleural effusion of the left side of the thorax associated with fever of 102° F. developed. Several aspirations followed but tuberculosis was not demonstrated by guinea pig inoculations. In a month's time this illness had abated, the pleural effusion was absorbed completely and for the following year his health and activities were normal. At the expiration of this year massive pericardial effusion occurred, the liver was enlarged and tender, and some ascites was present. Guinea pig inoculations of aspirated pericardial fluid disclosed no evidence of tuberculosis. Several pericardial aspirations were required. Although gradual improvement followed in the next few months, the enlargement of the liver and ascites persisted and transient edema of the extremities and the sacrum appeared. Seven months before admission to the Clinic generalized edema was present in the morning and disappeared during the day. This phenomenon ceased spontaneously and did not recur. For four months before admission, the ascites was more persistent and abdominal paracentesis was required with increasing frequency despite fairly effective diuresis produced by intravenous injections of mercurial diuretics.

Examination revealed prominent pulsations of the veins of the neck, a regular pulse with a rate of 100 beats per minute and blood pressure of 108 mm. of mercury systolic and 70 mm. diastolic. The heart was not considered to be enlarged on percussion and no murmurs were heard. Moisture was present at the base of both lungs posteriorly and there was bilateral hydrothorax. Ascites was marked and the edge of the liver extended 6 cm. below the costal margin. Dusky discoloration and mild edema of the legs and sacrum were observed. On roentgenographic examination of the thorax the heart was

considered to be moderately enlarged but no deposits of calcium could be demonstrated in the pericardium. The only significant urinary finding was the presence of a moderate albuminuria. Studies of renal function, serum proteins, the albumin-globulin ratio and tests on the blood yielded normal findings.

On June 3, 1939, partial pericardiectomy and partial epicardiectomy were performed under cyclopropane anesthesia with oxygen and helium. The usual circular incision was made over the pericardial region and the cartilages and about 3 cm. of the third, fourth and fifth ribs were removed. The entire pericardium was dense and firmly adherent to the epicardium. It was difficult to obtain a line of cleavage between the epicardium and the myocardium and in some regions a small portion of the fibrosed epicardium had to be left to protect the coronary vessels especially along the interventricular sulcus. There were two separate and distinct layers; the pericardium was thick and fibrous and the epicardium in some regions was even thicker than the pericardium to which it had become firmly adherent. Both the epicardium and pericardium were removed from the left ventricle first, then around the apex of the heart where it was very firmly adherent and along the right ventricle. The firm adhesions of the right ventricle to the diaphragm were then separated. The pericardium was partially separated from the right auricle and down to the entrance of the inferior vena cava. The operation was difficult because of the firm fixation of the adhesions of the epicardium to the heart muscle; in some places the epicardium had to be separated from the pericardium before it could be separated from the myocardium. The most dense adhesions were over the anterior longitudinal sulcus, the apex of the heart and between the right ventricle and the diaphragm. About half of the pericardium and approximately a third of the epicardium were removed which together with the separation of the adhesions to the diaphragm freed about three-fourths of the myocardium. Although there were dense adhesions over the left auricle, no attempt was made to remove the posterior portion of the pericardium in that region. Microscopic study of the tissue revealed chronic fibrous pericarditis.

Because of the difficulty and extent of the operative procedure a transfusion of blood was given during the operation. The patient's immediate reaction following the operation was satisfactory and his progress continued satisfactory until the tenth day when he began to have a fever of 100.5° F. At this time right pleural effusion developed which required aspirations. The effusion continued for four weeks. Following this the patient had a variable fever ranging from 99 to 102.5. This could be accounted for only by a small draining sinus in the wound which was still discharging a small amount of seropurulent material at the time of his dismissal. On the eighteenth day after operation abdominal paracentesis was done with removal of 2,500 c.c. of fluid. The patient's postoperative course was prolonged owing to the low urinary output, the protracted febrile reaction and pleural effusion. At the time of his dismissal the venous pressure in the right brachial vein was 260 mm. of water which indicated that inflow stasis of the heart was not relieved and renal excretion suffered in consequence from the impairment of the general circulation.

In a letter received from the patient six months after operation, he stated that the sinus at the site of the wound continued to drain. He was able to be up and about daily but found it necessary to have injections of mercurial

diuretics at intervals of eight days. His symptoms have not been entirely relieved and he has been advised to return for exploration of the sinus.

CASE 6.—A man, aged 43, complained of swelling of the abdomen and legs and of dyspnea on admission to the Clinic. Edema of the legs was noted ten months before admission and ascites occurred three months later. His health had been good until thirteen years prior to his present illness at which time he had had pneumonia complicated by empyema for which surgical drainage of the right pleural cavity was necessary. It was suspected that pericarditis was present at this illness but two attempts at pericardial paracentesis did not reveal any fluid or exudate. While the patient was still in the hospital, generalized edema developed. He remained in the hospital for three months and was given digitalis. Gradual improvement occurred although the edema of the legs never disappeared completely after this sickness. He worked at heavy labor for the next seven years in spite of being somewhat dyspneic. He suffered from bilateral pneumonia three years prior to his admission to the Clinic. He made a good recovery from this and continued to work until the onset of his present complaint.

Examination at the Clinic revealed marked cyanosis and some distention of the veins of the neck. The pulse was of small volume, totally irregular in rhythm and the rate of 60 per minute was accounted for by previous digitalis therapy. The blood pressure measured 110 mm. of mercury systolic and 65 mm. diastolic. The heart was not found to be enlarged on percussion, the tones were faint and a faint blowing systolic murmur was heard at the apex. The teleroentgenogram revealed only slight increase in the size of the heart and evidence of calcification at the lower left border. Moderate albuminuria was present and the specific gravity of the urine was 1.032. The value for blood urea was 32 mg. per 100 c.c. The bromosulfalein test of liver function revealed retention of dye grade 2, but the concentration of bilirubin in the serum was normal. The patient was not anemic and serologic tests for syphilis gave negative results. Abdominal paracentesis, administration of ammonium nitrate and salyrgan and the restriction of the intake of water and salt greatly improved the patient's condition and he was allowed to go home to follow a similar program. He forced himself to work at his occupation as a trucker despite his edema.

The patient was readmitted to the Clinic because of dyspnea which occurred on the slightest exertion. At this admission the dyspnea was more evident, the distention of the veins of the neck, more marked, the ascites, much greater and a large umbilical hernia had developed. Otherwise the findings on physical examination and the laboratory studies were not significantly different from those at the time of his previous admission.

On October 20, 1937, partial pericardiectomy was performed with partial removal of the pericardium from the left and right ventricles. Cyclopropane anesthesia was used. The usual curved incision was made; the cartilages, 1 inch (2.5 cm.) and about a third of the left border of the sternum were removed. The mediastinal structures and sternum were firmly adherent to the pericardium. The pericardium revealed marked calcification varying from 1 to 5 mm. in thickness. This deposit of calcium which was in one firm mass made a solid encasement of the heart and had invaded the pericardial attachment to the diaphragm. It was difficult to break through this calcified shell which was fully 0.5 cm. thick over the left ventricle. It was cut through with a bone cutter and a line of cleavage was found between the pericardium and the heart muscle of the left ventricle. The myocardium was carefully

dissected from the deposit of calcium. The left ventricle was freed first, then the right ventricle and then as much as possible of the right auricle. The deposit of calcium had to be removed in segments to avoid injury to the myocardium because it was almost as solid as bone. Posteriorly over the left auricle the calcium thinned out into a fibrous membrane. No attempt was made to remove this portion of the constricting pericardium. The left ventricle was completely freed up to the auriculoventricular sulcus. The right ventricle was separated from the calcified pericardium and from the diaphragm. It was impossible to remove large plaques of calcium which were firmly incorporated in the diaphragm. An attempt was made to remove some of the larger deposits of calcium over the right auricle but some of them had to be left in situ. The pericardium was separated as much as possible around the orifice of the vena cava. Although most of the calcified portion of the pericardium was removed from the ventricle, it was impossible to remove the calcium which was embedded in the diaphragm as well as that implanted in the right auricle and orifice of the vena cava. From this standpoint the operation was not satisfactory. The pericardium was drained with two Penrose drains. Microscopic study of the tissue removed revealed chronic pericarditis with thickened, fibrous and markedly calcareous walls.

The patient had considerable reaction following the operation and was placed immediately in oxygen. His postoperative course was stormy and complicated by a confusional state which could only be accounted for by impaired renal and hepatic function. His temperature was normal until the tenth day when he began running a slight fever and there was drainage of plaques of calcium and semipurulent material from the wound. Culture of the material drained yielded Streptococcus viridans. He was dismissed two months after operation.

The patient returned to the Clinic about one and a half years later. The wound had continued to drain and he had been unable to take much exercise because of dyspnea. In spite of restriction of the intake of salt and water, and therapy which had included digitalis, ammonium nitrate and salyrgan it was necessary for him to have abdominal paracentesis at intervals of ten to twelve weeks. Because of the continued drainage from the wound the sinus tract was excised and several large plaques of calcium were removed from the deep posterior portion of the sinus tract. This improved the type of drainage but failed to stop it entirely. Letters from the patient two years after operation indicated that the dropsical condition although markedly improved by the operation was practically unchanged from the time of his last visit.

CASE 7.—A frail appearing boy, aged 12 years, complained of swelling of the abdomen on admission to the Clinic on Sept. 28, 1936. As early as 1931 cyanosis had been observed after swimming and a diagnosis of congenital heart disease was made that year and again in 1932. The abdomen had been large for years but suddenly in 1933 it grew larger, the liver was found to be enlarged and episodes of cyanosis and dyspnea increased in frequency and severity. Abdominal paracentesis first was done in February, 1934. By November edema became generalized including the face in its distribution. Anorexia was the only symptom of digestive disturbance. The history of childhood infections included measles in infancy, pertussis at 4 and chickenpox at 5.

Physical examination revealed a moderate amount of cyanosis of the lips and fingers and a mild degree of icterus. The patient was emaciated. His height was 56¼ inches (138 cm.), his normal weight was 78 pounds (35.4

Kg.) and his weight at admission was 83 pounds (37.6 Kg.). The latter was accounted for by ascites and edema of legs, back and chest. The pulse was 100 and the rhythm was regular. The blood pressure was 96 mm. of mercury systolic and 54 mm. diastolic. The heart was not enlarged on percussion and no murmurs were heard on auscultation. The teleroentgenogram revealed thickened pleura and possible fluid at the base of the right lung and no evidence of cardiac enlargement. The urine contained a trace of albumin and the blood count, concentration of blood urea, blood sugar and serum bilirubin were within normal limits. The bromsulfalein test of hepatic function revealed retention of dye grade 2. The value for serum protein was 3.99 Gm. per 100 c.c. but the albumin-globulin ratio was normal. A diagnosis of Pick's syndrome was suggested but it was concluded that cirrhosis of the liver was present and a Talma-Morison operation was performed. No improvement followed this procedure and the patient was transferred to the cardiac service under the care of one of us (A. R. B.). After reconsideration of the clinical history and findings it was concluded that a diagnosis of chronic constrictive pericarditis was warranted and pericardiectomy was advised. The patient was in poor condition owing to the long duration of the disease and to the degree of emaciation present. Attempts to secure diuresis with diuretics met with little success and after abdominal paracentesis it was considered that no further treatment would prepare the patient any better for the surgical procedure.

On Oct. 31, 1936, partial pericardiectomy was performed under cyclopropane and oxygen anesthesia. The usual curved incision was made with resection of the cartilage and segments of the third, fourth, fifth and sixth ribs and removal of the left half of the sternum with the rongeur. The pleura was very adherent to the pericardium and was carefully dissected lateral to the phrenic nerve. The pericardium was hard and firm and deposits of calcium could be felt by palpation on the anterior surface. On incising the anterior wall of the pericardium, it was found to be 5 to 8 mm. in thickness. There were practically two layers of pericardium with a thin, granular deposit of calcium extending from the base of the heart and involving the diaphragmatic portion of the pericardium. The calcium was found to be in irregular, thin sheets with sharp edges. These sharp edges made careful removal necessary in order to avoid injuring the myocardium. The pericardium was separated from over the left ventricle first, and then from over the right ventricle. A few of the larger plaques were dissected from over the right auricle and around the vena cava. It was necessary to leave most of the calcium in this region. It was impossible completely to separate the upper portion of the right ventricle from the diaphragm as the plaques of calcium had penetrated the heart muscle on one side and the diaphragm below. As the muscle was very friable, considerable hemorrhage occurred on attempting to remove the calcium from this region. It was possible to separate about half of the attachment of the right ventricle to the diaphragm but it was impossible completely to separate the apex of the heart. About a third of the pericardial sac was removed. The wound was closed with two small Penrose drains. A transfusion of blood was given while the patient was on the operating table. Pathologic examination of the tissue removed revealed acute and chronic fibrous pericarditis with calcareous degeneration.

The operation was difficult and tedious and was unsatisfactory because of the inability to separate the calcium from the diaphragmatic portion of the pericardium, the right ventricle and apex of the heart. The patient was placed

immediately in the oxygen chamber. His immediate convalescence was more satisfactory than was expected. His condition remained fairly good for the first twenty-four hours after which the blood pressure decreased. His temperature rose to 104° F. in the afternoon of the second day and the respiratory difficulty gradually increased. The patient died on the fourth day after operation. The immediate cause of death was pneumonia; the contributing factors were hypertrophy and chronic passive congestion of the liver and ascites.

CASE 8.—A well-developed man, aged 41, registered at the Clinic on Aug. 5, 1938, complaining of orthopnea, paroxysmal dyspnea and edema of the ankles. These symptoms had been present for four months. Additional symptoms included mild precordial pain on effort, easy fatigability, anorexia and belching.

The patient had contracted gonorrhea in 1913 and urethral discharge continued until 1919. In 1915 arthritis in the knees and ankles had occurred. In 1919 the patient was bedfast two weeks with cough, fever and pain in the right shoulder but recovery from this was apparently complete. For the next five years his health was excellent and he was able to swim six miles at a time. In 1924 cardiac enlargement was found on roentgenoscopic examination of the thorax. A roentgenogram of the chest in 1929 revealed calcification of the pericardium but aside from occasional dyspnea on exertion there were no indications that cardiac function was impaired.

On physical examination at the Clinic in 1938 pulsation of the veins of the neck was observed but there was no evidence of cyanosis. The pulse was regular and its rate was 76. The blood pressure was 100 mm. of mercury systolic and 66 mm. diastolic. The left border of the heart was at the outer limits of normal; a slapping type of the apical impulse and a suggestion of a thrill were present. The heart sounds were muffled and there was a rough presystolic rumble at the apex. The teleroentgenogram of the thorax disclosed a maximal cardiac width of 15 cm. as compared to a transverse diameter of the thorax of 32 cm. and also revealed marked calcification of the pericardium. There was no evident ascites, the edge of the liver was felt 2.5 cm. below the costal margin and the edema of the ankles was graded 2. The urine contained a small amount of albumin, the value for blood urea was 32 mg. per 100 c.c.; examination of the blood revealed hypochromic macrocytic anemia of mild degree. Serologic tests for syphilis gave negative results.

On Aug. 20, 1938, partial parietal pericardiectomy was performed under cyclopropane anesthesia with the addition of oxygen and helium. The usual curved incision was made over the precordial region with resection of the cartilages and segments of the third, fourth, fifth and sixth ribs. The pleura was adherent to the pericardium and was separated laterally to the level of the left phrenic nerve. The pericardium adhered firmly to the sternum and to the mediastinal structures. Many large vessels required ligation during its separation from the sternum. The entire pericardium adhered firmly to the heart muscle; the anterior surface was hard because of large calcareous deposits. There was little calcium between the visceral and parietal pericardium at the apex of the heart but the two layers were firmly adherent. There was a heavy layer of calcium on the diaphragmatic portion of the parietal pericardium at the apex which bound the right ventricle firmly to the diaphragm over at least half of its surface. There was no definite line of cleavage between the visceral and parietal pericardium and the separation

had to be made with the aid of scissors and knife. The calcium was more than 1 cm. thick in places along the longitudinal and auriculoventricular sulci, on the ventricles and around the orifice of the vena cava. The pericardium was separated from the left ventricle first and then from the right ventricle freeing the apex of the heart. In the longitudinal sulcus there were two layers of cleavage. The dissection was made toward this sulcus in two directions to avoid injury to the vessels; however, one of the veins in the sulcus was injured and bleeding was controlled by suturing the fascial layer at this point. The greatest difficulty was encountered in removing the visceral pericardium from the apex of the heart as well as from the diaphragm because in this region the calcium was very thick. Around the orifice of the vena cava there was not only calcium but also some soft putty-like material which was removed with a curet. The heart enlarged markedly after the removal of the calcium and was very friable showing evidence of marked myocardial degeneration. Large plaques of calcium could be felt in the left auriculoventricular groove and extended to the region of the left auricle, but no attempt was made to remove the calcium from this region. A transfusion of blood was given during the operation. Pathologic examination of the tissue removed disclosed chronic pericarditis with marked fibrosis.

The operative removal of the constricting pericardium and calcium was not complete in this case although a fairly large amount of the encasing membrane and calcium was removed. It was not advisable to do a more extensive operation because of the friability of the myocardium. The patient was placed in an oxygen tent immediately after the operation. His immediate postoperative condition was satisfactory. The second postoperative day he began to show signs of pulmonary congestion with increase in respiratory rate. This gradually increased and he died three days following the operation from bilateral bronchopneumonia with beginning formation of abscess. The contributing factors were cirrhosis of the liver and hypertrophy of the spleen and kidneys. Examination of the heart revealed considerable residual calcium in the pericardium and in the fold of the left ventricle close to the sulcus there was a large plaque of calcium which extended through the entire wall of the ventricle presenting in the ventricular chamber of the heart. This may have been the result of a previous cardiac infarct.

This proves the danger of attempting to remove deposits of calcium which involve the heart muscle. An attempt to remove this segment of calcium from the left ventricle would have resulted in a large opening into the ventricular cavity.

CASE 9.—A man, aged 27, stated that swelling of the legs and abdomen and breathlessness on exertion had been present for one year and these symptoms had grown progressively worse during that time. This illness had begun with a sore throat, attended by a high fever; he had been in bed for about six months. For two weeks he had severe pain in the left anterior and lower parts of the thorax. A diagnosis of pericarditis and myocarditis was made which accounted for four months of this confinement to bed. The pain was diagnosed as caused by pleurisy. For six months prior to admission he had been up and about but the legs and abdomen were swollen greatly and he passed a subnormal amount of rather dark urine. He had had rheumatic fever at ten years of age.

Examination of the patient at the Clinic disclosed slight cyanosis of the lips and ears and definite pulsations of the veins of the neck. The pulse was

98, its rhythm was regular and the systolic blood pressure was 115 mm. of mercury and the diastolic pressure 85 mm. The heart was found to be moderately enlarged on percussion, the edge of the liver extended three fingerbreadths below the right costal margin, the abdomen was distended with fluid and the legs were markedly swollen. In the teleroentgenogram of the thorax the heart shadow was enlarged and the shadow of the diaphragm was somewhat indistinct presumably owing to bilateral pleural effusion. Study of the urine, renal function, plasma chlorides, the carbon dioxide combining power of the plasma, the serum bilirubin and other tests on the blood revealed nothing abnormal.

On Aug. 1, 1938, partial pericardiectomy with removal of the constricting fibrous epicardium was performed under cyclopropane anesthesia with the addition of helium and oxygen. The usual circular incision was made in the left anterior chest wall. The cartilages and segments of the third, fourth, fifth and sixth ribs were removed. A small portion of the sternum was removed with the rongeur. On separating the adherent left mediastinal pleura from the pericardium the left pleural cavity was opened and about 1,000 c.c. of cloudy pleural effusion was aspirated. The opening in the pleura was closed. The pericardium was rounded and distended from pericardial effusion. On incising the pericardium it was found to be markedly thickened and there was a thick exudate adherent to its inner surface. There was about 500 c.c. of fluid in the pericardial cavity. A large amount of fibrin attached to both the heart and the pericardium gave them a bread-and-butter appearance. The largest mass of fibrin was posteriorly.

After removing the fibrin and fluid it was found that the epicardium was markedly thickened and formed a complete constricting membrane surrounding the myocardium. It was very difficult to find the line of cleavage between the myocardium and this adherent fibrous layer. It had to be carefully dissected with a knife or scissors. In some places it was as much as 0.5 cm. thick. The constricting tissue was removed segmentally from the left and right ventricles. After the fibrous constricting tissue was removed from the heart muscle, the amplitude of the heart beat as well as the size of the heart increased. It was necessary to leave a portion of this fibrous capsule along the anterior longitudinal sulcus as it was very adherent to the underlying coronary vessels, and to leave several patches of the thickened pericardium. These portions of fibrous tissue were cut in several directions, as in the Rentzeloff type of operation to permit expansion of the myocardium. The constrictive covering was removed from the greater part of both ventricles and a transfusion of blood was given during the procedure. Tissue from the pericardium revealed chronic fibrous tuberculosis microscopically.

The patient's immediate convalescence was satisfactory. The temperature and respiration remained normal for the first six days. On the seventh day he became irrational and the pulse rate increased. There had been considerable drainage from his wound from the onset. This became more marked and was of a typical tuberculous type. His temperature gradually rose and remained around 102° F. His mental state varied; on some days he would recognize persons about him but the greater part of the time he was irrational. He took food well until the week prior to his death. He gradually became worse from inanition and he died on the twenty-ninth postoperative day.

The operative procedure in this case from a surgical standpoint was unsatisfactory because the cardiac tamponade was due both to constriction

of the myocardium from the fibrous epicardium, pressure from the pleural effusion and the underlying etiologic factor which was tuberculosis. Such cases are not favorable for surgical intervention.

General Comment on Findings at Operation

The etiology of chronic constrictive pericarditis is obscure. It is generally agreed that rheumatic fever is not its basis. A history of rheumatic fever was obtained in only one case (case 9) and in this case pathologic study of the pericardial tissue removed at operation revealed that tuberculosis was the etiologic agent. In one other case (case 5) a history of contact with tuberculosis was established, but guinea pig inoculations, pathologic examination of tissue removed· bacterial stains and cultures did not demonstrate any evidence of tuberculosis. In two cases (2 and 6) the pericardial involvement had followed multiple attacks of pneumonia. In five of our cases there was either no history of significant antecedent infections or if any infection had occurred it was mild and gave no indication that it was attended or followed by involvement of the pericardium. Smears and cultures of material removed from the pericardium at operation were studied in all cases and yielded negative findings.

On physical examination cyanosis was observed in five of our cases and pulsating or distended veins of the neck were observed in eight. The blood pressure was uniformly low, the average systolic pressure was 106 mm. of mercury and the average diastolic pressure 71 mm. In cases of this type the pulse is likely to be fast and in our cases the average pulse rate was 87 beats per minute. In six cases the cardiac rhythm was regular, in two auricular fibrillation was present and in one the rhythm was interrupted by occasional premature contractions.

No murmurs were heard over the heart in seven cases; indefinite and non-diagnostic cardiac murmurs were heard in two. In six cases the heart was not enlarged on percussion or on roentgenographic examination. In two slight cardiac enlargement was demonstrated by roentgenographic methods. The heart shadow was definitely enlarged in one case (case 9) but this was accounted for by a large effusion in the pericardium. Roentgenographic examination of the heart revealed calcification of the pericardium in six cases. In an additional case calcification of the pericardium was found at operation. Special roentgenographic technic with a short exposure to minimize the movements of the heart probably would have demonstrated calcification of the pericardium in this instance.

The electrocardiographic findings in these cases were striking and suggested or strongly supported the diagnosis of chronic con-

strictive pericarditis. The electrocardiographic pattern which is most diagnostic is one in which the QRS complexes are of low voltage (below 5 mm.) and T waves are negative in all the standard leads. If the QRS voltage is low in only one lead that will usually be observed in lead I. The T waves in the standard leads may be of low voltage though upright, they may be iso-electric or may be inverted in only two leads. The Wolferth precordial lead is characterized by an exaggerated Q wave at the expense of an R wave of diminished amplitude. Similarly the R wave in leads IV R and IV F is disproportionately large as compared to the S wave. The T wave in the Wolferth precordial lead is upright in more than half the cases and it frequently arises from an R-T segment that is slightly elevated. The T wave in lead IV R is negative in about half the cases and it arises frequently from an S-T segment that is slightly depressed. The electrocardiograms change very little after operation in the cases in which the operation is not successful in relieving inflow stasis. In cases in which inflow stasis is relieved by operation the electrocardiogram tends to return to normal after operation though some trace of the original electrocardiographic abnormalities may persist for a long time in spite of the fact that normal cardiac function has been restored.

Studies of direct venous pressure were made by means of a water manometer in seven of our cases before operation. The venous pressures were elevated markedly in each case: the maximum pressure was 315 mm. of water, the minimal, 220 mm. and the average pressure, 265 mm. The venous pressure in case 1 measured 90 mm. of water six months after operation. In another case in which the clinical result was not entirely satisfactory, the venous pressure was 190 mm. of water fifty days after operation, as compared to a preoperative venous pressure of 282 mm. The venous pressure obtained a considerable period after operation is probably the most accurate index of the degree of relief of inflow stasis.

The arm-to-tongue circulation time, determined by the injection of dehydrocholic acid (decholin) into an antecubital vein, was determined in six cases before operation. In all but one case (case 4) it was found to be distinctly prolonged and in case 4 there is reason to doubt the accuracy of the determination. The average circulation time from arm to tongue was twenty-six seconds. In one case in which the clinical result of operation was excellent, the circulation time was thirty-seven seconds before operation and seventeen seconds six months after an operation. In another case (case 4) in which the clinical improvement was only fair the circulation time was twenty-five seconds fourteen months after operation.

Bromsulfalein tests of hepatic function were made in all cases at the time of the initial examination. In one case retention of dye was graded 4, in four cases graded 3, and in four graded 2. After treatment which included administration of mercurial diuretics and ammonium nitrate, abdominal paracentesis and intravenous infusions of solutions of glucose, the function of the liver was again determined in three cases just prior to operation. In the two cases in which retention of dye had been graded 3 at the initial determination, the preoperative retention was only graded 2 and in the third case it had dropped from grade 4 to grade 2. We feel that impairment of hepatic function plays a crucial role in the patient's postoperative reaction and that preoperative attempts to improve hepatic function are highly important. In the light of the newer knowledge of vitamins it seems worth while to include a large amount of the vitamin B complex in the preoperative preparation. In case 1 in which the clinical result was ideal, there was no retention of dye six months after operation. In case 2 in which the clinical result was also excellent, retention of dye, determined forty days after operation, had not changed from the preoperative level. In a second similar case (case 3) the retention of dye determined thirty days after operation was unchanged from the preoperative figure. This indicates that the hepatic function recovers slowly after an operation which is successful in relieving inflow stasis of the heart.

Surgical Treatment

Anesthesia.—A general anesthetic is advisable because the operation may require considerable time. This time factor is very tiring for the patient and if a general anesthetic were not employed, it would add considerable nervous strain which would interfere with the cardiac function. Furthermore if respiratory difficulties arise, it is advisable to have the patient under complete control. The anesthetic agent in our cases was administered with a closed mask but an intratracheal tube was available for use if necessary at any moment. The anesthetic agent employed was cyclopropane in all cases. We prefer cyclopropane as the anesthetic agent because it is a distinct advantage in cardiac cases to be able to administer a high percentage of oxygen to prevent cyanosis during the operation. The pleura was opened in three of our cases and closed by suture. There were no harmful effects and it was not necessary to introduce the intratracheal tube.

Operative procedures.—The approach to the pericardium and heart was through the left side of the thoracic wall in all cases. A curved incision was made from the sixth rib below and a little to the left of the nipple line; it extended along the sixth cartilage to

the midportion of the sternum, along the sternum as high as the third rib and then along the third rib to the nipple line. The operative technic is as follows:

The pectoralis major muscle is dissected from the chest wall and reflected with the skin flap. The costal cartilages of the sixth, fifth, fourth and in some instances the third ribs are resected subchondrally and subperiosteally from the sternum to and including 3 to 5 cm. of the corresponding ribs. The intercostal muscle flap is then cut from the left border of the sternum and the internal mammary vessels are ligated both above and below. After dissecting the inner muscle flap, the pericardial attachments to the sternum are separated and the left half of the sternum is resected with the rongeur. The left pleura then is dissected carefully from the pericardium laterally as far as the left phrenic nerve. This pleura is often very adherent particularly when the pericardium is calcified and openings may be made into the pleura during the dissection. These openings in the pleura are immediately closed with catgut. It is important to have adequate exposure of the entire left surface of the pericardium and the number of ribs necessary to resect varies in different individuals depending on the vertical diameter of the heart and its pericardial attachments.

After reflecting the pleura from the left and lateral folds of the pericardium, the resection of the pericardium is started over the left ventricle. The left ventricle is freed first to establish better function and disposal of the blood before freeing the right side of the heart. The freeing of the right side of the heart permits a greater inflow of blood into the right ventricle and if the myocardium is badly damaged, this increased pressure may result in a dilatation of the right side of the heart unless the transmission of this blood is facilitated by freeing the left ventricle first. The point of incision into the pericardial scar should be well over on the left ventricle to avoid injury to the anterior descending portions of the left coronary vessels. The site of election depends to some extent on the type of scar and accessibility of the myocardium. Great care should be exercised to ascertain the actual point of fusion of the pericardial scar and the myocardium before removal of the pericardium is started. There is often a great variation particularly when deposits of calcium are present. In some instances the calcified tissue may extend into the heart muscle and great care is required in removal to avoid serious injury to the heart muscle. During the removal of this scar these deposits of calcium should be left attached to the reflected pericardium. In some regions, particularly around the deposits of calcium, the fusion plane is not uniform and the dissection must be carried around the deposit in slightly

different planes before it can be removed by sharp dissection. When the calcium invades the heart muscle, no attempt should be made to remove it as the tissue involved may extend entirely through the heart muscle as occurred in case 8.

There is some difference of opinion as to the amount of pericardium it is necessary or advisable to remove as well as to the amount of scar to be separated from the heart muscle and from the orifice of the inferior vena cava. We believe it is advisable to remove as much of the pericardial scar as possible from the ventricles, the right auricle and orifice of the inferior vena cava and to separate the scar from the diaphragmatic attachments of the right ventricle. It is particularly important to separate the apex of the heart and this should be done early in the operation.

In many instances the calcium is granular and is often piled up around the orifice of the inferior vena cava, around the right auricle and right auriculoventricular sulcus. This was particularly true in four cases of our series. The calcium was removed with a curet and then some of the underlying fibrous scar was incised. Because this was done in cases 1, 2 and 3 in which the best surgical results were obtained, we believe the removal of this calcium and scar around the right auricle, sulcus and orifice of the vena cava may be an important factor. We do not believe it is advisable to attempt to remove a solid calcified scar attached to the right auricle. Removal of the scar from the left auricle was not attempted in any case. It is advisable to remove the scar in a segmental manner and in instances in which the scar tissue seems to have penetrated into the heart muscle, small segments of scar were left attached to the myocardium rather than run the risk of damage to the muscle or to the coronary vessels.

During the course of the removal of the pericardial tissue the pulse rate often becomes very rapid and irregular owing to the external stimulus to the heart muscle. When the pulse becomes very irregular, it is important to cease the operation and frequent rest periods and applications of sponges moistened with warm saline solution permit the heart to regain or at least partially regain its former rhythm.

In our cases the wound was closed in two layers with drainage at the lower angle in all cases. The inner layers of intercostal muscle and perichondrium were sutured to the posterior sternal fold with interrupted catgut sutures. The skin and outer layers of muscle were closed with interrupted dermal sutures. At the lower end of the wound just to the left of the sternum the inner layer was not sutured for a space of from 2 to 3 cm. and two small Penrose

drains were placed between the diaphragm and the heart. This permitted the immediate removal of the serous exudate from the precordial space. The drains were removed from 36 to 48 hours after operation. There was always a varying amount of serous exudate following removal of the adhesions, even though great care was taken to prevent trauma to the tissues as well as to attain accurate hemostasis. If this fluid is permitted to accumulate in the precordial space, it may interfere with the function of the heart which is already laboring under the increased stress resulting from the operation. If it is not permitted to drain externally, the fluid may rupture into the pleural space and interfere with respiratory function. When granular deposits of calcium are scattered throughout the pericardium, it is particularly advisable not to have this material drain into the pleural cavity because of the risk of pleural irritation and empyema.

Postoperative Care

All patients were placed immediately in oxygen cabinets or tents and were left for varying periods depending on their progress. This is one of the most important considerations in postoperative treatment, for it not only prevents cyanosis but aids in relieving stress on the heart. Other postoperative means are directed toward removal of body fluids and aiding the function of the liver. Intake of fluid is limited. Measures are similar to those utilized preoperatively for similar purposes.

Results

It is impossible to draw any definite conclusions from such a relatively small group of cases, particularly as the condition was unusually severe. In seven cases extensive calcification was associated with the fibrous constricting pericardium. Of the two cases of fibrous pericarditis without calcification, one (case 9) was proved to be due to tuberculosis and in the other (case 5) tuberculosis was suspected although not proved.

Three patients were cured, three were improved by operation and three died 3, 4 and 29 days after operation. In attempting to evaluate results of this small group the first important consideration is the selection of cases for operation which entails the determination of the amount of myocardial and hepatic damage which has taken place. This is often difficult to evaluate clinically and it is impossible to determine the amount of re-establishment of function and regeneration which will take place after operation.

In only one of the three cases (cases 7, 8 and 9) in which death followed operation could any degree of improvement be expected

in the light of the operative findings. This was in case 7. It is probable that the damage to the liver was the most important factor in the patient's inability to recover following operation. In cases 8 and 9 an appreciable improvement was improbable, for in case 8 the myocardial degeneration with deposits of calcium extended entirely through the heart muscle making improvement of cardiac function impossible and in case 9 the underlying etiologic factor was tuberculosis.

In the three cases (cases 4, 5 and 6) in which improvement occurred, the operative results from a surgical standpoint were disappointing in one (case 4) as satisfactory freeing of the ventricles was obtained and there did not seem to be a great amount of myocardial or hepatic damage. It is possible that this patient may improve more after a longer period has elapsed. If this improvement does not occur, further surgical intervention should be considered because constriction of the heart may have recurred and it may be necessary to attempt to remove more of the constricting fibrous tissue around the orifice of the vena cava and the right auricle. In the other two cases (cases 5 and 6) the patients did not present the true picture of Pick's disease. The operative results were probably as good as could be expected in cases of this type. In one (case 5), tuberculosis was suspected although not proved; the patient presented an unusual history with intermittent fever and although there was a definite constriction of the pericardium this same condition has continued in less degree since operation. In case 6 the symptoms were of fifteen years' duration. There was a very dense stony covering over the myocardium which was degenerated markedly. The patient's condition has been considerably improved by operation. Because the myocardial degeneration made it impossible to remove all of the calcium, it seems to us that he had obtained as satisfactory a result as can be expected and that no further surgical intervention should be attempted.

The three patients (cases 1, 2 and 3) who were considered cured, presented the typical picture of Pick's disease, and all had extensive calcification of the pericardium with constriction of the heart muscle. The excellent results obtained in these three cases after adequate removal of the constricting pericardium, exemplify the capability of the heart and liver to re-establish function if the damage has not been too great. In all three the myocardium was separated satisfactorily from the surrounding attachments.

Conclusions

The important factors in the operative treatment of constricting pericardium are wide resection of the pericardium, the complete freeing of the apex of the heart, separation of the right ventricle from the diaphragm and when deposits of calcium are present, removal of the calcium as much as possible from the right auricle, the auriculoventricular sulcus and from around the orifice of the vena cava.

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OBSTRUCTIVE JAUNDICE: DIAGNOSIS AND TREATMENT

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THE clinical differentiation of obstructive jaundice from that due to a toxic hepatitis is at times most difficult despite a detailed history, a careful physical examination, and the use of many types of diagnostic procedures. It is most important to attempt to distinguish jaundice due to a toxic hepatitis from other types of obstructive jaundice, since an operation in a case of toxic or infectious hepatic jaundice is not only futile but may be very dangerous. Before attempting such a differential diagnosis it is necessary that we understand the mechanism of production and the significance of jaundice.

Of the several theories to explain the different types of jaundice, that of McNee[1] seems to be the most logical. Bilirubin is formed by the reticulo-endothelial system from the hemoglobin of destroyed erythrocytes through an intermediary product, hematin, by the process of hydrolysis, but is normally excreted only by the liver. As bilirubin is brought to the liver, or formed in the Kupffer cells, it passes through the polygonal cells from the hepatic vascular capillaries (sinusoids) into the fine bile ducts. When bilirubin reaches the intestine it is converted into urobilinogen by the action of intestinal bacteria. Most of the bile pigment is excreted in the stool as urobilinogen or as its oxidation product urobilin. That part of urobilinogen which is not excreted in the stool is absorbed by the intestinal mucosa and returned to the liver by way of the portal circulation. Most of the urobilinogen which reaches the liver is re-excreted, and the remaining small amount enters the hepatic vein and is excreted in the urine as urobilinogen. In one type of jaundice there is an obstruction in the bile ducts which may be either extrahepatic or intrahepatic. Since there is no outlet for the bile pigment it passes back through the polygonal cells into the sinusoids whence it enters the hepatic vein gaining direct access to the general circulation with the production of jaundice.

A second type of jaundice, toxic and infectious, may be produced in the absence of an obstruction of the bile ducts because of direct injury to the hepatic cells which may be unable to excrete the bilirubin brought to them from the reticulo-endothelial system. Thus, a certain amount of bilirubin, depending on the degree of hepatic damage, fails to pass through the polygonal cells to the bile capillaries and is returned to the general circulation by way of the hepatic vein.

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A third type of jaundice, the so-called hemolytic type, is produced by the excessive destruction of erythrocytes. Such destruction of red blood cells produces more bilirubin than the liver cells can excrete, the excess bilirubin spilling over into the general circulation.

Unfortunately more than one type of mechanism may simultaneously play a role in the production of the jaundice, which may make it a very complicated problem.

The common causes of obstructive jaundice are mechanical blocking of the biliary system, usually from acute or chronic inflammation associated with calculi, and tumors of the ducts or the head of the pancreas. The site of obstruction may be in the intrahepatic bile ducts because of the presence of edema and exudate produced by cholangitis. The bile capillaries may be also compressed by edema of the hepatic cells in hepatitis.

The diagnosis of *obstructive jaundice due to a stone in the common duct* is usually made from the history of repeated attacks of biliary colic, vomiting, and chills and fever associated with a history suggesting cholelithiasis. Practically all cases have had previous biliary tract disease, and colic is present in about 80 to 90 per cent of them. Pronounced spontaneous vomiting is very suggestive of a cystic duct or common duct stone. Chills and fever are present in about one third of the cases, and are due to an associated cholangitis. About one third of the cases give a history of having had one or more attacks of jaundice at a previous time. When a history of intermittent jaundice can be obtained it is of much value in differentiating between obstruction due to a stone and that due to malignant disease. Loss of weight is more suggestive of malignancy, but it can be present in patients with obstruction due to stone who have pronounced vomiting. Common duct stone is generally thought to occur more frequently in women than men. Cutler and Zollinger[2] found the ratio to be 9 to 1 in their series. Meyer and Steigmann,[3] however, favored the men 2 to 1 in a smaller group of cases.

Carcinoma of the head of the pancreas, along with common duct stone, comprises one of the two most common causes of obstructive jaundice. When jaundice is caused by malignant disease the symptoms usually develop insidiously, in marked contrast to the acute symptoms of stone in the common duct. In malignant disease the jaundice is progressive instead of intermittent. Pain is much less pronounced than in calculous obstruction, although the majority of patients have distress of varying severity in the epigastrium, right upper quadrant, or back. Weight loss occurs in a high percentage of cases. Carcinoma of the head of the pancreas is much more common in men than women, and like carcinoma elsewhere there is an

increasing severity of symptoms. Physical examination may be of much value in differentiating between obstructive jaundice due to common duct stone and that due to carcinoma of the head of the pancreas. In the former there is usually tenderness in the epigastrium and the right upper quadrant, but rarely an enlarged palpable gallbladder. However, in about 50 per cent of the cases of carcinoma of the head of the pancreas a large palpable gallbladder is present, and there is frequently found enlargement of the liver. In the malignant cases an irregular hard mass may be palpated in the epigastrium but this is probably not found often enough to be of much value.

Obstructive jaundice may be produced by large metastatic glands in the portal fissures, and by possible direct extension to the extrahepatic ducts from a carcinoma of the gallbladder. Such a process may be accompanied by a past history of biliary tract disease, but unlike common duct stone the symptoms are progressive without tendency to remission, and the condition occurs usually at a later period in life (average age around 60 years). Primary carcinoma of the bile ducts usually occurs at a still later period in life, and produces early intense jaundice due to complete obstruction.

Jaundice due to a stricture of extrahepatic bile ducts may be caused by injury or inflammation due to a stone, but is usually caused by technical difficulties or errors at the time of operation. If the common duct has been completely ligated severe persistent jaundice immediately follows the operation. A history of profuse discharge of bile following cholecystectomy continuing somewhat intermittently for a long period of time is strongly suggestive of a stricture due to an injury to the duct at the time of operation. In such a case jaundice may not appear until months after the operation, and after the biliary fistula has closed. Jaundice from such a cause is usually painless, of varying intensity, and is accompanied by intermittent signs of cholangitis, such as chills and fever, due to infection resulting from poor bile drainage.

In the hepatocellular jaundice, there are two clinical types. One group consists of catarrhal jaundice, infectious hepatitis, spirochetal icterus, and sepsis, all of which may be placed under the heading of infectious hepatic jaundice. A second group is classed as toxic jaundice and includes the various types of poisoning such as cinchophen, arsphenamine, chloroform, lead, mercury, phenylhydrazine and carbon tetrachloride. Jaundice following overdose of x-ray, as well as jaundice occurring in toxemias of pregnancy, belong in this group.

A carefully taken history should do much toward making a correct diagnosis in the latter group. Too often, however, the neces-

sary information is not obtained until after the patient has been needlessly operated upon. This is particularly true in cases of acute hepatitis and acute yellow atrophy following the administration of drugs containing cinchophen.

Acute catarrhal icterus most commonly occurs during the first and second decades of life and usually follows an acute gastrointestinal upset or an acute respiratory infection. Patients with this condition are usually not very sick. Jaundice is frequently mild, and may not even be noticed by the patient. Colic is not present, and nausea and vomiting are not conspicuous features. Patients usually complain of malaise, anorexia, and mental depression. The temperature is not elevated or only slightly so, and the pulse shows little change from the normal except in those cases with deep jaundice, when there is a bradycardia. The icterus clears up spontaneously after a period of ten days to eight weeks. On physical examination a tender slightly enlarged liver may be found, and occasionally a distended gallbladder may be palpated. In the unusual case occurring after the fourth decade of life, accompanied by intense jaundice, a palpable gallbladder, and loss in weight, it may be very difficult to differentiate it from a carcinoma of the head of the pancreas.

Hemolytic jaundice is observed clinically in familial hemolytic icterus, acquired hemolytic jaundice, icterus neonatorum, malaria, sickle-cell anemia, pernicious anemia, concealed hemorrhage, hemolytic post-transfusion reactions, and septicemia produced by hemolytic bacteria. There is usually no difficulty in differentiating this type of jaundice from the other two types. In familial hemolytic icterus and acquired hemolytic jaundice, the spleen is always enlarged and can usually be palpated. There is mild icterus and pain is slight. There may be mild weakness and decreased resistance to various infections. It should be remembered that the presence of gallstones is not an infrequent finding in hemolytic jaundice. Probably the most important diagnostic feature of the familial type is the increased fragility of the red blood cells.[4] The stools instead of being clay-colored contain more bile pigment than is normally found. Little bilirubin is found in the urine although there is a large amount of urobilinogen present.

The laboratory findings are of great value in making a differential diagnosis of the three types of jaundice. To determine the patency of the biliary passages a chemical test for the presence of bile in the stools is a necessity. The color of the stools may be of little value, since the excessive amount of fat present in the stools of jaundiced patients may mask the color of the bile pigment. If after stool examination there is doubt as to the presence of bile, duodenal intubation should be done.

The van den Bergh[5] reaction is the most accurate method for the quantitative determination of bilirubin in the blood. It was formerly thought that the direct reaction was pathognomonic of extrahepatic biliary obstruction, but this view is no longer held. The direct reaction does occur characteristically in the obstructive type of jaundice, but is frequently obtained in toxic or infectious jaundice. The direct reaction occurs in the latter type of jaundice when the condition has progressed to the degree of marked hepatic cell damage with edema obstructing the bile capillaries. In hemolytic jaundice the indirect van den Bergh reaction is always obtained, provided there is no associated hepatic damage.[6]

In the last few years many liver function tests have been devised to determine whether or not jaundice in a given case is hepatogenous or obstructive in origin. These tests are based on the fact that early in the course of biliary obstruction the blood may contain substances which are forced into circulation, and that the liver itself is very little disturbed early in the course of obstructive jaundice, while in hepatogenous jaundice there are often early and serious disturbances of some of its essential functions. It is generally agreed that hepatic damage always follows obstruction of the biliary passages, and this damage may appear early, greatly confusing the clinical picture. Then, too, the obstruction may be incomplete and of such short duration that the composition of the blood is very little changed. Also, functional disturbances in jaundice of liver origin may be difficult to detect when the initial injury is quickly repaired producing little evidence of dysfunction. Therefore, the time element must be kept in mind in using the various tests. Definite results may be obtained in uncomplicated types of jaundice of short duration, but the chance for error is great in the cases of chronic jaundice of long duration.

Probably the most important functional test from the standpoint of differential diagnosis is the quantitative determination of fecal and urinary urobilinogen.[7] When no urobilinogen can be found in the urine of a jaundiced patient, the diagnosis of complete extrahepatic biliary obstruction should be made. Obviously in such a case urobilinogen will also be absent from the stools. Such a finding indicates neoplastic obstruction. If there is partial extrahepatic biliary obstruction, such as that due to a stone, fecal urobilinogen will be decreased, but rarely completely absent. In such a case urinary urobilinogen will be decreased unless the partial obstruction is accompanied by hepatic damage when an increased amount of urobilinogen is present. In infectious and toxic jaundice, typified by catarrhal jaundice and arsphenamine poisoning, there is usually a preliminary rise in urinary urobilinogen. At the height of the disease complete intrahepatic obstruction may develop so that fecal

and urinary urobilinogen completely disappears. The obstructive stage, however, is only transient, lasting usually three to four days or, at the most, seven days. This stage is accompanied by the highest level of hyperbilirubinemia. With the spontaneous relief of the intrahepatic obstruction, urobilinogen reappears in the urine in large amounts. The amount of urobilinogen decreases as the acute inflammation of the hepatic cells subsides. Therefore, to differentiate between acute intrahepatic jaundice and obstructive jaundice of neoplastic character it is essential that repeated urobilinogen determinations be made for several days.

Other tests which may be of value are elimination of dyes,[8] plasma cholesterol,[9] galactose tolerance,[10] hippuric acid,[11] serum phosphatase activity,[12] and serum proteins.[13]

There are other procedures[14] which may be helpful in making the correct diagnosis. A blood amylase determination may be valuable early in the disease when pancreatitis is associated with a common duct stone. An ordinary x-ray film of the abdomen may reveal stones in the region of the gallbladder or a primary shadow furnishing valuable information. Cholecystography, however, is of little value in cases of jaundice. A gastrointestinal x-ray study may be of much value in making a diagnosis of obstructive jaundice due to a tumor. For obvious reasons, a routine blood count, urine analysis, and blood Wassermann should be done.

When, by careful observation and study of the jaundiced patient, we have determined that the cause of icterus is due to extrahepatic biliary obstruction, the treatment is obviously surgical. These patients are poor surgical risks due to the liver damage that is invariably present and a marked hemorrhagic tendency that is so often present. The results of surgical treatment of jaundice have improved a great deal recently, due to more adequate preparation of the patient and the use of recent developments which have been made in the prevention and treatment of complications. Selection of the proper time for surgical intervention is of no little importance. The patient should be in the best possible condition when operation is done. There is seldom an indication for an emergency operation in this type of case. If icterus is subsiding, operation should be delayed. In an occasional case of obstruction of the common duct due to calculus in which there is constant or increasing icterus associated with signs of acute cholangitis, such as chills and fever, it is advisable to delay operation no longer than is necessary properly to adjust the fluid balance.

It is of great importance in the preparation of the jaundiced patient to obtain a glycogen reserve in the liver in order to prevent and combat hepatic insufficiency, as shown by Graham[15] in 1915.

This is accomplished by giving a high carbohydrate diet for several days prior to the operation. It is of equal importance to continue glucose therapy postoperatively.

It is also of major importance to correct any deficiency in blood prothrombin before operating on a case of obstructive jaundice. Until two years ago postoperative hemorrhage in these cases was greatly feared. Prior to this time it was thought that the intravenous administration of calcium chloride[16] aided in hastening the coagulation of blood, and it was given routinely to combat the hemorrhagic tendency. In 1935, however, Quick, Stanley-Brown, and Bancroft[17] cited experimental evidence to prove that calcium, as well as fibrinogen and thromboplastin, are present in normal amounts in the blood of jaundiced patients, and that the only substance deficient in quantity for the proper coagulation of blood is prothrombin.

In 1934 Dam[18] found that a hemorrhagic tendency developed in chicks which were fed on a diet deficient in certain fat-soluble compounds, but adequate in vitamins A, B_1, B_2, C and D, and in total fat and cholesterol. He demonstrated that the hemorrhagic tendency was promptly corrected by the administration of a substance obtained from hog-liver fat and from alfalfa which he called the "coagulation vitamin" or "Vitamin K."[19,20]

If vitamin K is not tolerated by mouth or if the prothrombin clotting time is greatly prolonged, it is advisable to administer vitamin K and bile salts by means of duodenal tube. The mixture should be given slowly requiring 30 to 60 minutes for its administration. Usually 10,000 units of vitamin K[22] and 2 grams of bile salts will lower the prothrombin clotting time to normal.[23] However, in cases of actual hemorrhage postoperatively, it may be necessary to repeat the procedure.

It must be remembered that in patients with badly damaged liver it is not infrequent to find no response to the administration of vitamin K.[24,25] The lack of response in these cases is due to the inability of the damaged liver to utilize vitamin K in the formation of prothrombin. Blood transfusions offer the best means of increasing the prothrombin content of the blood in such patients. All patients with obstructive jaundice should be cross-matched for transfusion before operation, since blood transfusion is still the quickest way of raising the blood prothrombin level, and may be a life saving measure in the emergency of hemorrhage. Only fresh blood should be used since the prothrombin concentration of blood decreases rapidly on standing.[26]

In the few cases of obstructive jaundice in which I have had an opportunity to administer vitamin K the results have been excellent.

One patient, a 40-year old white woman, on whom I operated recently for a stricture of the common bile duct, had had intermittent jaundice for over three years, and had been operated on three years previously for the same condition, at which time there was marked bleeding from the operative wound and from the urinary bladder, requiring frequent blood transfusions. The prothrombin clotting time, by the method of Ziffren, Owen, Hoffman and Smith,[21] was 40 per cent of normal. After five days of vitamin K therapy preoperatively, the prothrombin clotting time was 80 per cent of normal. At operation, which consisted of relieving the obstruction by means of a T-tube, there was no excessive bleeding, and there was no postoperative hemorrhage.

Experimental work indicates that protein and vitamin C[30, 31] are of importance in the process of healing. Thompson Ravdin, and Frank[32] found that dogs fed on a low protein diet showed impaired healing, and that disruption of abdominal wounds occurred in 72 per cent. Harvey and Howes[33] found an acceleration of fibroplastic proliferation in dogs on a high protein diet.

Obstructive jaundice due to carcinoma of the head of the pancreas is no longer considered a hopeless condition. Whipple[37] in 1935 successfully resected the head of the pancreas for carcinoma which was completely obstructing the common bile duct. Since that time, several cases of radical resection of the head of the pancreas have been reported. Some of these cases have lived over three years without evidence of recurrence. All patients with obstructive jaundice due to malignancy should have the benefit of surgery. It is true that exploration frequently reveals that the carcinoma has extended beyond the limits of surgical excision, but even these patients can be made more comfortable by relieving the biliary obstruction by doing a cholecystogastrostomy or cholecystojejunostomy. The radical operation for resection of the head of the pancreas should be done in two stages, because of the increased mortality associated with the one-stage operation.

Because of the occurrence of cholecystitis and cholangitis, Whipple[38] for the past two years has been ligating the common duct below the cystic duct, and performing an "antecolic cholecystojejunostomy on the Roux principle of anastomosing the distal cut end of the jejunum to the fundus of the gallbladder, with an end-to-side anastomosis of the proximal cut end of the jejunum to the side of the jejunum 10 cm. below the cholecystojejunostomy." About three weeks after the first-stage operation, a gastrojejunostomy and excision of the second portion of the duodenum and the head of the pancreas are done. Cholangitis has not occurred so frequently following this plan of procedure.

The saying that "no man is infallible in the diagnosis of obstructive jaundice" still holds true. All of us, however, should strive, by constant study, observation and research, to make fewer errors in diagnosis, and to approach nearer to that goal of perfection by which we will be able more adequately to treat patients with obstructive jaundice.

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THE PREVENTION OF WOUND DISRUPTION

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THE postoperative disruption of an abdominal wound is a tragic and deplorable complication.

Hartzell and Winfield, of Wayne University, reported in January, 1939, an analysis of 1,458 cases of wound disruption and reviewed 116 papers on this subject.

As the literature grows the factors responsible would appear to become more and more complicated and mysterious. Age, anemia, malnutrition, diabetes, malignancy, vitamin C deficiency, intra-abdominal distention, the vertical incision, the use of catgut instead of silk, the omental wedge, are all factors which have been suggested by various authors as playing a part in the causation of this disaster.

In 1933, at the November meeting of the New York Surgical Society, the subject became the topic of a general symposium. Dr. Frank Meleney reported 55 cases which had occurred at the Presbyterian hospital in the previous 8 years, an incidence of 1 per cent with a mortality of 44 per cent. He stated that the actual incidence might be 2 per cent.

Dr. Ralph Colp reported 29 cases from a study of 2,750 consecutive laparotomies at Mount Sinai Hospital, an incidence of 0.9 per cent with a mortality of 28 per cent.

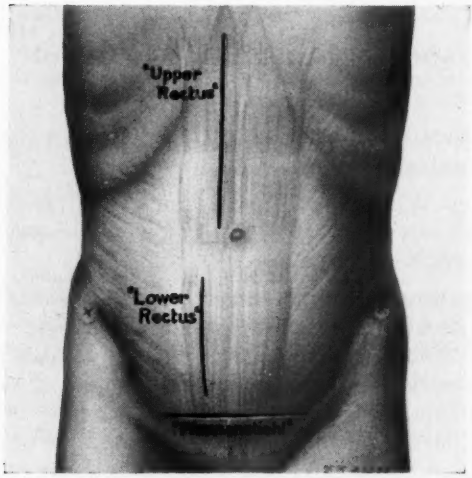
Dr. Roderick V. Grace reported from the First Surgical Division at Bellevue Hospital, 46 cases occurring during the previous 15 years with a mortality of 29 per cent.

Dr. William C. White reported from Roosevelt Hospital 30 cases with a mortality of 53 per cent. At this Hospital there is an average of from one to four cases a year, but in 1931, out of 406 vertical abdominal incisions, there were 7 disruptions.

Dr. Charles Gordon Heyd reported from the Post-Graduate Hospital a series of 2,145 personal laparotomies, with 4 disruptions, and a mortality of 25 per cent. In 1932 there were 1,000 laparotomies performed at the same Hospital by a group of surgeons with 4 disruptions.

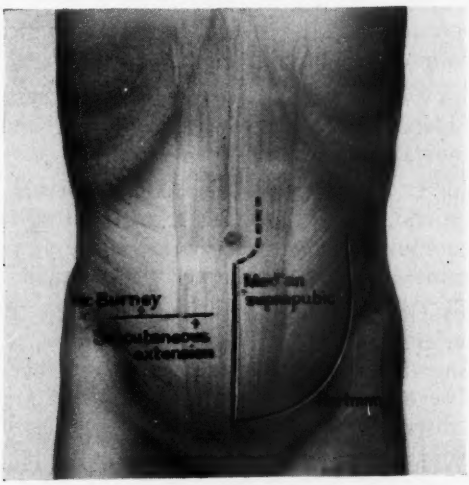
Dr. Allen Whipple, in 1933, at the Presbyterian Hospital in New York, reported his advocacy of silk in the repair of clean abdominal wounds. He thereby hoped to reduce the incidence of infection and disruption. Dr. Whipple and Dr. Meleney and their coworkers

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.



1. Location upper and lower rectus and Pfannenstiel incisions.

at the Presbyterian Hospital have made an unusual contribution in calling attention to this question of wound infection and disruption. Their advocacy of silk has resulted in bringing about a great improvement in the catgut supplied by practically all the manufacturers



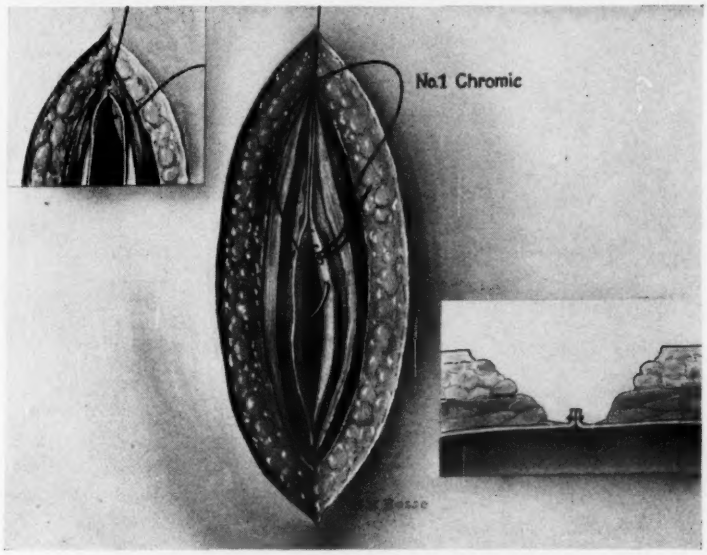
2. Location McBurney, median suprapubic and Hartmann incisions.

and has emphasized the desirability of using finer suture material and a more gentle technic.

In approaching a consideration of this subject, one is at once confronted with the problem of wound healing in general. What

factors promote healing and what retard the process? What part does the physical condition of the patient play in the healing of his wound, and what can the surgeon contribute by his particular technic?

It has long been recognized that malnutrition, anemia, cachexia, diabetes and other constitutional states retard the healing process.



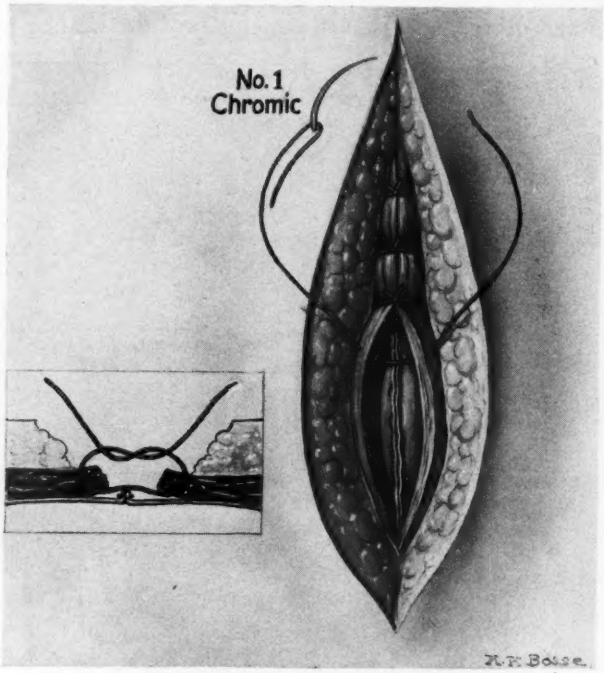
3. Vertical incision: peritoneal closure.

The recent studies in vitamin C deficiency by Bartlett, Jones and Ryan, of Boston, place a new emphasis on this factor in relation to surgical patients.

Their studies indicate that a fasting blood plasma level of under 0.5 mg. is definitely abnormal, that the normal saturation state, with respect to vitamin C will show a fasting blood plasma level of about 1.3 mg. per 100 c.c. of plasma. An increase in the vitamin C intake fails to raise the level and a large proportion of the excess vitamin appears in the urine. The range between 0.5 mg. per 100 c.c. and saturation must be considered normal, although many falling within this range show varying degrees of depletion. This is an important study and may prove helpful in evaluating the probability of delayed wound healing.

This picture of vitamin C deficiency is not new. Richard Walter's account of Lord Anson's Voyage Around the World in 1749 vividly describes the effect of scurvy among the crew:

Scars of wounds that had been for many years healed, were forced open, one of the invalids, who had been wounded fifty years before had continued well for a great number of years past, yet, on his being attacked by the scurvy, his wounds in the progress of the disease broke out afresh and appeared as though they had never been healed.

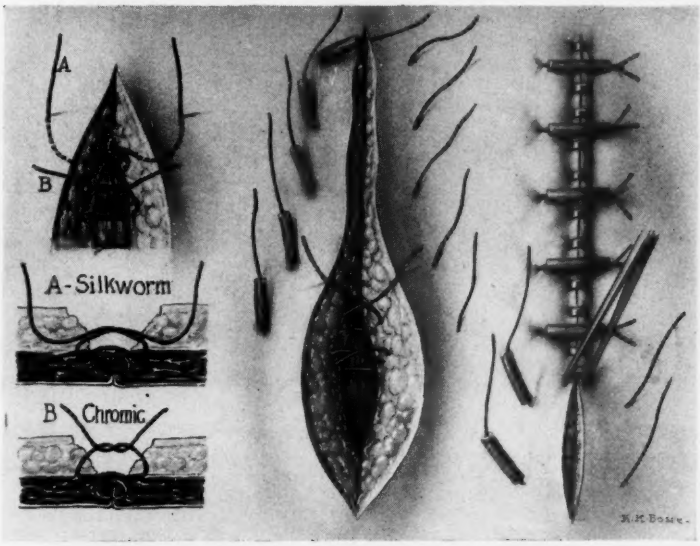


4. Vertical incision: muscle closure.

The effect of hypoproteinemia on wound disruption was reported in 1938 by Thompson, Ravdin and Frank of the University of Pennsylvania. This is also a very interesting study, fortified by animal experiments, proving that a deficiency in protein causes tissue edema and retards wound healing. The postoperative administration of large amounts of sodium chloride increases the tissue edema incident to a low level of serum protein and further retards the healing process. The use of lyophile plasma is suggested by this group to correct hypoproteinemia and to prevent wound disruption.

I have had no personal experience with lyophile plasma but have depended on whole blood transfusion where indicated. This has become an increasingly simple procedure since the establishment of our blood bank which is enthusiastically recommended and seems to be the answer to the transfusion problem.

Catgut allergy is another factor which is of interest, Kraissl, Kestos and Cimiotti, of the Presbyterian Hospital, New York, reported in 1938, an interesting piece of experimental work on this question. Twenty-five per cent of animals sensitized to plain catgut disrupted their wounds, as against a control of 100 per cent healing



5. Vertical incision: anterior rectus sheath closure.

in nonsensitized animals. Three hundred thirty-two patients were tested for sensitivity to plain catgut extract, chromic catgut extract and chromic acid. Fourteen per cent gave a positive reaction to one or more of these solutions.

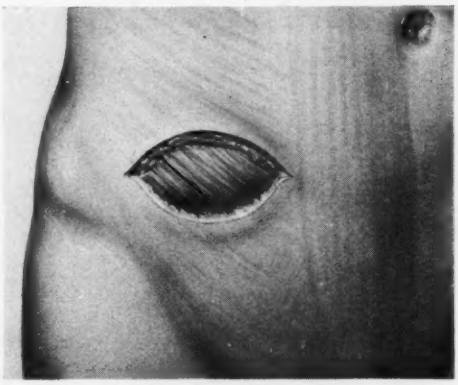
In refutation of this work on catgut allergy, Kenneth L. Pickrell, in the Johns Hopkins Bulletin of March 1939, reports that

> in the human being no evidence of sensitivity could be demonstrated following the disruption of three surgical incisions that had been closed with catgut. In 100 clinic cases in which catgut had been used, tests for sensitivity to catgut extract were carried out pre-operatively and then one month following operation. In no case was sensitivity demonstrated. One can conclude that, although catgut, like any other suture material, acts as a foreign body and causes a slight leucocytic response, it does not act as an antigen to induce the hypersensitive state.

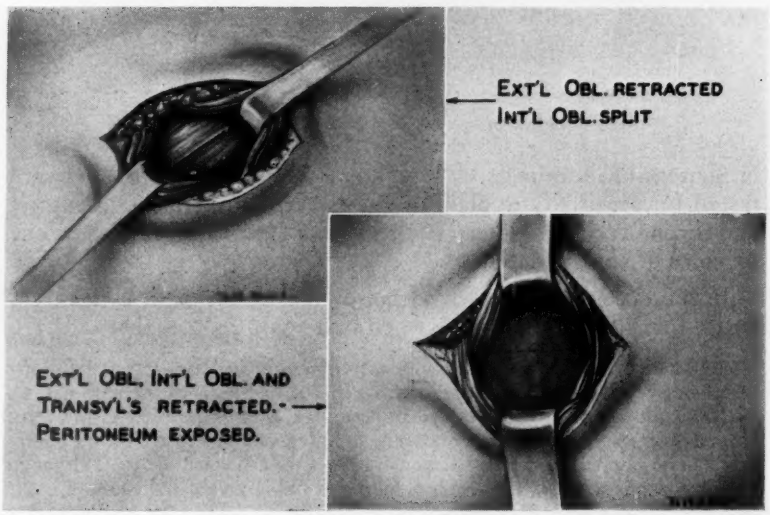
Historically, we might carry the discussion back to the days of Hippocrates, who practiced and taught 400 years before the birth of Christ. Hippocrates emphasized, in his treatise on the treatment of wounds, the advantages of asepsis, the importance of close ap-

proximation of wound edges and the desirability of rest and immobilization to facilitate the healing process.

Pare, in 1564, substituted the ligature for the cautery and in his teachings so emphasized the healing powers of nature that today his statue bears the inscription, "Je le pansay, Dieu le guarit."

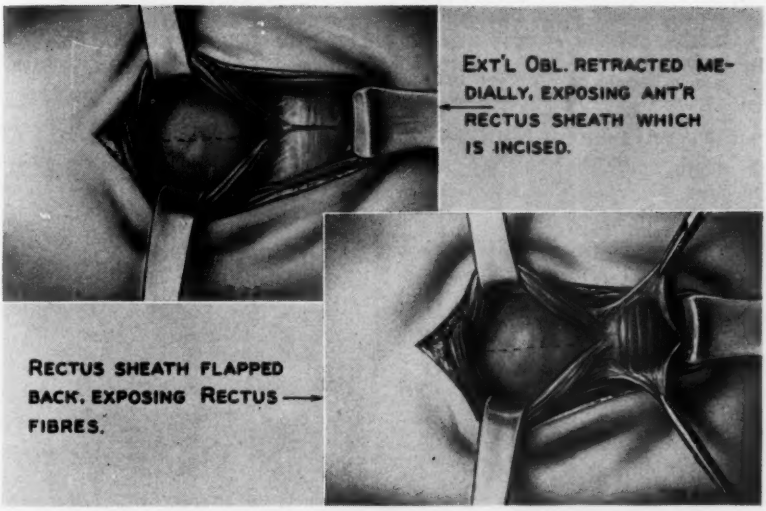


6. Modified McBurney incision.

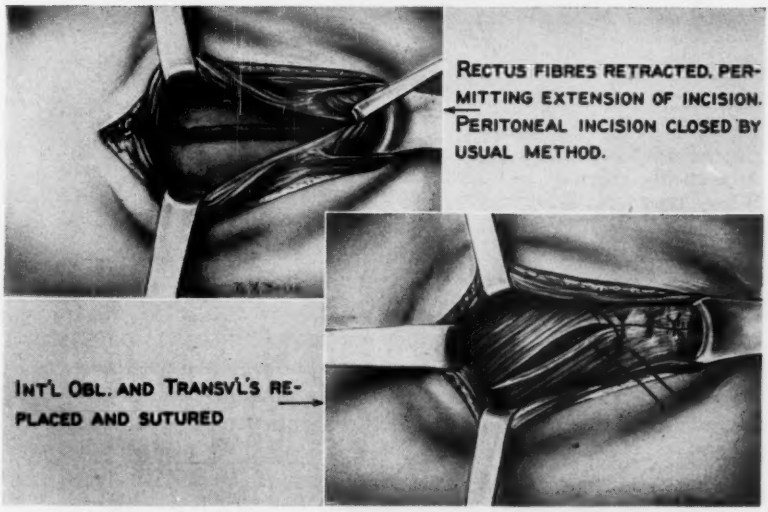


7. McBurney incision: retraction.

However, the purpose of this presentation is to call attention to a simple method of abdominal wound closure which has been used over a number of years in a consecutive series of 2,148 laparotomies and has been complicated by only two disruptions. Perhaps one of


EXT'L OBL. RETRACTED MEDIALLY, EXPOSING ANT'R RECTUS SHEATH WHICH IS INCISED.
RECTUS SHEATH FLAPPED BACK, EXPOSING RECTUS FIBRES.

8. McBurney incision: deep retraction.


RECTUS FIBRES RETRACTED, PERMITTING EXTENSION OF INCISION. PERITONEAL INCISION CLOSED BY USUAL METHOD.
INT'L OBL. AND TRANSV'L'S REPLACED AND SUTURED

9. McBurney incision: closure peritoneum and muscle.

these should be discarded because, through an error in the supply of suture material, the supposed silkworm gut was not furnished.

This patient, a man of 60, was operated on for a carcinoma of the stomach. A subtotal gastrectomy was followed by an uneventful convalescence. On the fifth day, at the time of the first dressing,

TABLE 1

A STATISTICAL STUDY OF 2,148 LAPAROTOMIES, DRAINED AND NOT DRAINED, GROUPED ACCORDING TO THE PRIMARY CONDITION FOR WHICH THE OPERATION WAS PERFORMED AND ACCORDING TO THE TYPE OF INCISION EMPLOYED.

	Appendicitis Acute	Appendicitis Chronic	Biliary Tract	Stomach	Large or Small Bowel	Gynecologic	Miscellaneous	Total
Modified McBurney								
Drain.....	91	1	...	...	3	1	...	96
No Drain...	601	107	...	...	5	6	2	721
Upper Rectus								
Drain.....	...	...	167	9	...	...	2	178
No drain...	...	...	14	87	4	...	3	108
Lower Rectus								
Drain.....	48	...	...	...	53	11	1	113
No drain...	122	81	...	...	50	27	9	289
Median								
Drain.....	3	...	5	2	20	75	3	108
No drain...	7	6	1	8	25	315	32	394
Transverse, Pfannenstiel & Miscellaneous								
Drain.....	...	...	...	...	...	...	16	16
No drain...	...	7	...	...	3	111	4	125
Total cases ...	872	202	187	106	163	546	72	2,148

TABLE 2

CLASSIFICATION AS TO AGE GROUPS

	0-10 Years	11-50 Years	51-90 Years
Appendicitis, acute	115	706	51
Appendicitis, chronic	10	178	14
Biliary tract	...	122	65
Stomach	1	68	37
Large and small bowel	4	78	81
Gynecological	...	491	55
Miscellaneous	6	50	16
Total	136	1,693	318

TABLE 3

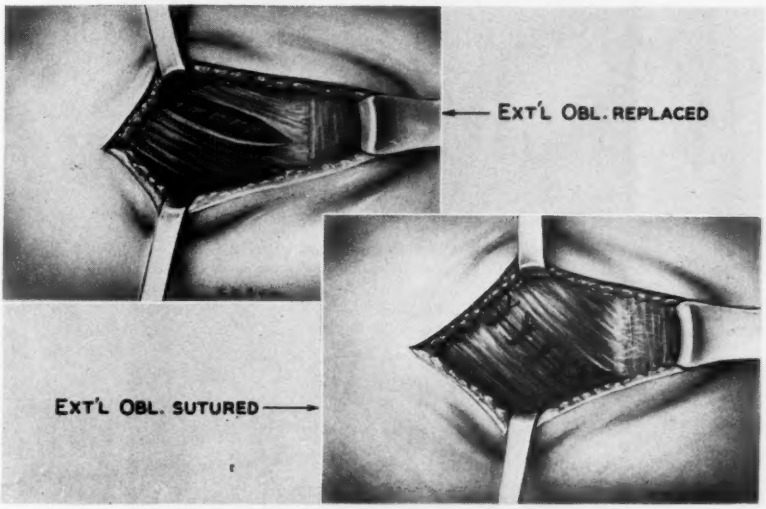
AVERAGE NUMBER HOSPITAL DAYS

Appendicitis, acute	13 days
Appendicitis, chronic	15 days
Biliary tract	21 days
Stomach	20 days
Large and small bowel	26 days
Gynecological	16 days
Miscellaneous	31 days

This grouping would seem to represent our best available evidence of post-operative morbidity and wound healing.

all of the supposed silkworm gut retention sutures were found disintegrated. A few hours later the wound burst open. It was resutured with the routine technic and healed by primary union. The patient lived for two years and died of metastases.

If this disruption is eliminated because of the fact that the routine suture material was, in error, not used, we may say that there is

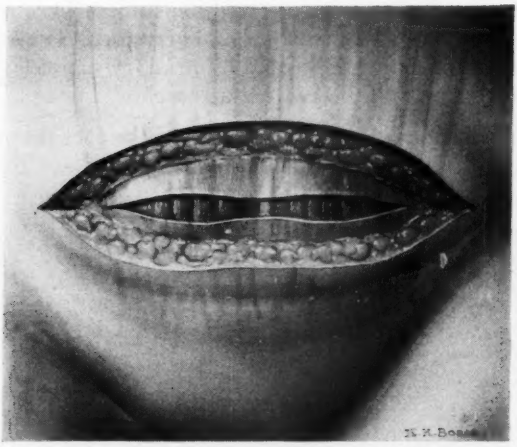


10. McBurney incision: closure external oblique.

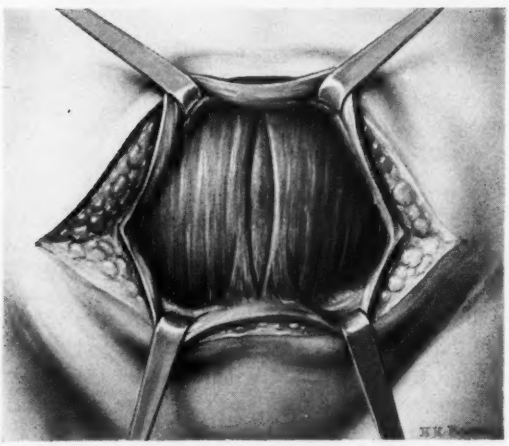
only one disruption to be reported. This was a desperately sick man of 54 who had contracted amebic dysentery at the World's Fair in Chicago. This was associated with an extensive ulcerative colitis and a perforation of the cecum with a large pericecal and pelvic abscess, subsequently followed by a subhepatic and a large intrahepatic abscess. Following several operations to drain the cecal and pelvic abscesses, an attempt was made, through a right upper rectus incision, to drain his liver and subhepatic abscesses. There was an extensive infection of the wound, hemorrhage from the liver and on the seventh day the wound disrupted. This was closed with interrupted silkworm gut through and through sutures. Two days later the patient died.

The 2,148 consecutive laparotomies which I am reporting were performed with a variety of incisions.

In the upper abdomen the ordinary vertical rectus incision was usually made; for exploratory procedures a right or left lower rectus



11. Transverse Pfannenstiel incision.

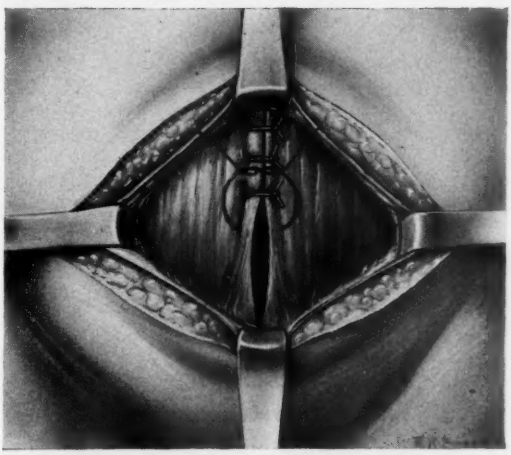


12. Transverse Pfannenstiel incision: retraction.

incision; for gynecologic operations a median suprapubic incision or a transverse Pfannenstiel incision and for some colon resections, a transverse suprapubic incision extending obliquely upward through the left iliac fossa. For the most part these were the routine incisions employed in this series of cases. If they had disrupted with the 2 per cent frequency reported in some clinics, there would have resulted over 40 disruptions, instead of the 2 reported.

In the presence of infection I have drained these wounds with soft rubber tubes or cigarette drains. These drains have been

13. Transverse Pfannenstiel incision: peritoneal closure.

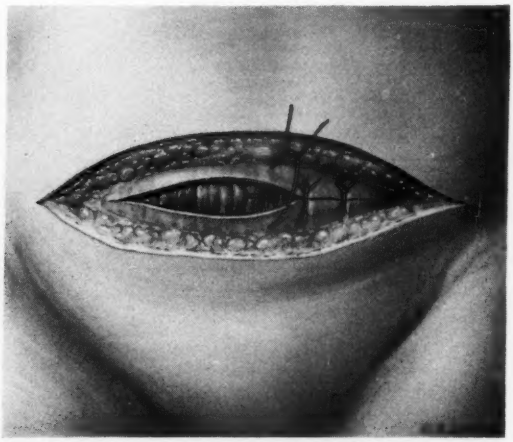


14. Transverse Pfannenstiel incision: muscle closure.

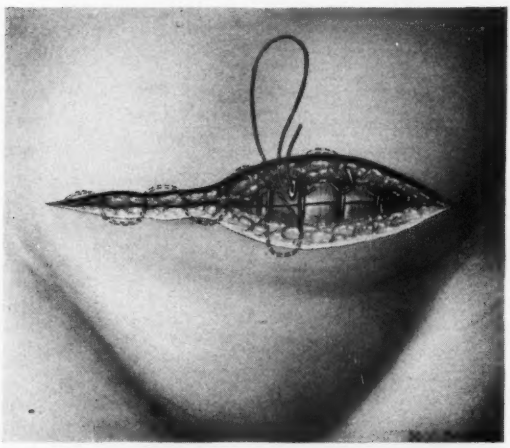
brought out through the wounds and not through counter stab wounds. Certainly, neither the type of incision nor the method of drainage has offered any special safeguard against disruption.

I believe that the method of closure employed in this series of cases is the factor which has practically eliminated the disrupted wound.

All vertical incisions are closed by the careful approximation of anatomic structures. Tension is avoided and gentleness in the



15. Transverse Pfannenstiel incision: rectus sheath closure.



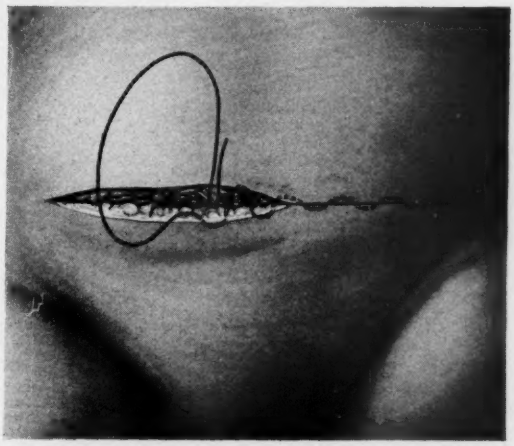
16. Transverse Pfannenstiel incision: deep fascia closure.

handling of tissue is stressed. The peritoneal layer is closed by a continuous "in and out" suture of chromic, everting the edges and passing at right angles instead of parallel to the fibers of the transversalis fascia and peritoneum. The separated muscle fibers are approximated with interrupted chromic, the anterior rectus sheath is closed with alternating chromic and figure of eight silkworm gut tension sutures which, when tied over pieces of rubber tubing obliterate dead spaces and supplement a careful hemostasis. The skin edges are approximated with dermal or Michel clips. This technic

was devised to give maximum support with a minimum amount of suture material to be absorbed by the tissues or left in the wound.

The skin sutures or clips are removed routinely on the fifth day but the tension silkworm gut sutures are never removed before the tenth day and sometimes not before the fourteenth day or later.

The McBurney incision is closed in layers with fine chromic, without the tension sutures.



17. Transverse Pfannenstiel incision: subcuticular closure.

The transverse Pfannenstiel incision is closed in layers with a continuous chromic suture for the peritoneum, interrupted chromic sutures for the other layers, without tension sutures. The skin is approximated with a subcuticular suture of plain catgut.

The use of silk versus catgut in the closure of abdominal wounds has been a controversial subject for years. Today we are witnessing the "renaissance of silk" and one who uses catgut is apt to be challenged.

In 1882 or 1883 Dr. Halsted substituted silk for catgut in all his surgical work, and in 1889, when Johns Hopkins Hospital was opened he outlined a "fine silk technic for the control of hemorrhage and the closure of wounds."

At the time, however, when Dr. Halsted was advocating the change from catgut to silk, it is only fair to consider a general picture of the surgical technic of that period. On October 14, 1884, Dr. Halsted made the following remarks at a meeting of the New York Surgical Society.

It would be difficult, in a given case, to know just what to attribute a bad result to, with the imperfect antiseptic technic at present existing even in our

best hospitals in New York. Trained nurses with long sleeves and hands uninspected are allowed to pass and hold dry sponges. I have repeatedly seen ligatures handed to the operator from the mouth of the interne and seldom failed to find instruments, especially artery clamps, which had been sufficiently cleaned. In some hospitals the preparation of the catgut is entrusted to the apothecary, and even if it is prepared by the interne, it is not a guarantee that it had been properly, even if conscientiously, done.

TABLE 4

MORTALITY ACCORDING TO DISEASE GROUPS

	Total	Died	Mortality
Appendicitis, acute	872	16	1.8
Appendicitis, chronic	202	0	...
Biliary tract	187	18	9.6
Stomach	106	21	20.0
Large and small bowel	163	40	24.3
Gynecological	546	9	1.6
Miscellaneous	72	9	11.1
Total	2,148	113	5.2

TABLE 5

MORTALITY ACCORDING TO CAUSE OF DEATH

113 Deaths in 2,148 Cases or a General Mortality of 5.2 Per Cent	
Postoperative shock	22
Peritonitis	18
Toxemia of obstruction	16
Cardiac	15
Postoperative pneumonia	12
Progress of disease	12
Hemorrhage	6
Embolism	5
Cholemia	2
Suppression of urine	2
Thyroid crisis	2
Wound disruption (peritonitis)	1
Total	113

TABLE 6

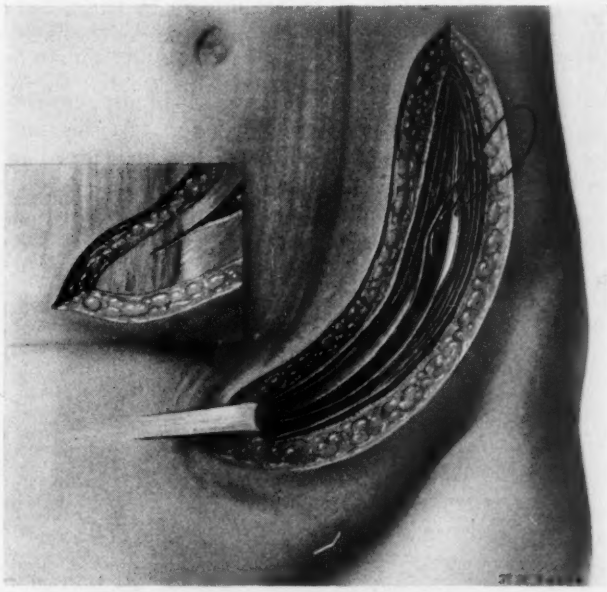
MISCELLANEOUS MORTALITY STATISTICS

38 OF THE 113 MORTALITIES OR 33.6 PER CENT WERE MALIGNANCIES

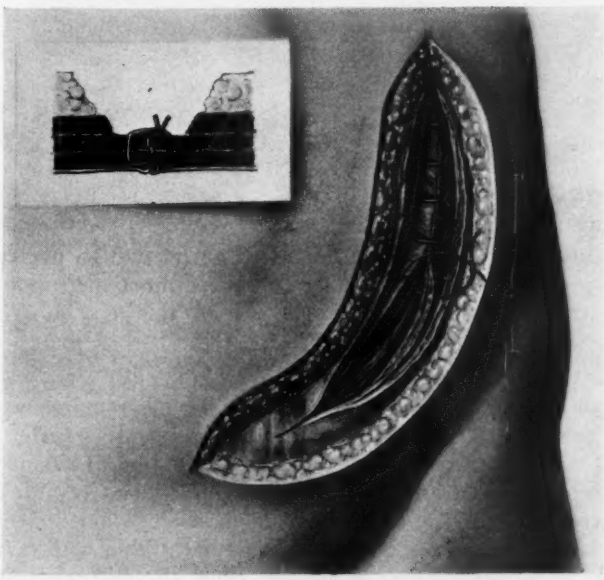
19 of the 113 Cases were over 70 years old

Post-op. Day of Death	Number Cases	Per Cent Mortality
1-5th day	76	67.3
6-10th day	21	18.5
13-377th day	16	14.2

There has certainly been a great improvement in the quality of catgut since the Halsted area. In the past decade we have seen the



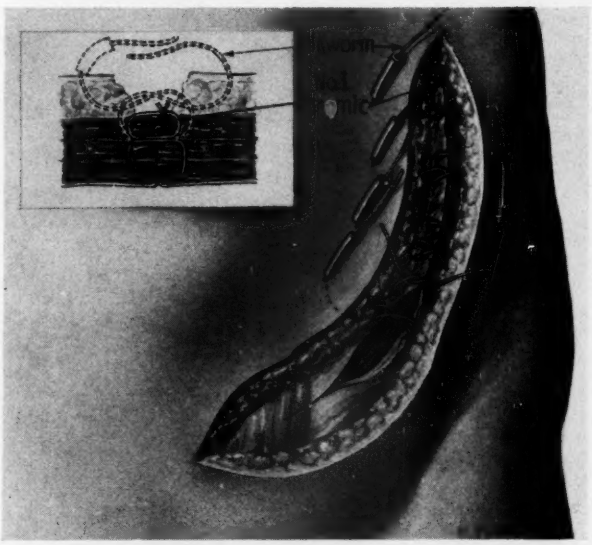
18. Suprapubic-iliac fossa incision: peritoneal closure.



19. Suprapubic-iliac fossa incision: muscle closure.

standardization and control of the sterilizing of catgut, great improvement in tensile strength and, recently, the development of the extremely fine, even to No. 00000, suture material.

In quoting Dr. Halsted on this question, one must remember that Dr. Halsted died in 1922, eighteen years ago. At the time of his death he was using the technic which he had developed in 1889. Should the silk technic, as advocated over fifty years ago by Dr. Halsted, because of the unsatisfactory catgut of those days, be adopted in our hospital practice today?



20. Suprapubic-iliac fossa incision: external oblique closure.

The advocates of silk claim, quite properly, that its use requires greater gentleness in the handling of tissues, more meticulous care, finer instruments and neater work. All of this may be true, but there is no reason why the same respect for tissues should not be observed, irrespective of the suture material employed.

Catgut has many advantages over silk. Its use requires no ritual. It may be tied with reasonable facility and speed. Its greater tensile strength is a protection against the early separation of wound edges. The complication of a slight infection in the wound resulting from the disease found at operation, or from some other source, may prove a great nuisance if the wound has been filled with many silk ligatures. The extra time required to close a wound with silk prolongs the anesthesia and increases the risk of postoperative pulmo-

nary and circulatory complications. This prolongation of operative time also predisposes to infection by air contamination. The great number of fine silk sutures required to approximate a wound with any degree of safety would seem likely to predispose to interference with the blood supply of the tissues in the wound and retard the normal healing process. The difference in the cost between silk and catgut may be offset by the cost of prolonged anesthesia and operating room service.

I have had little personal experience with silk but my experience with the use of catgut and silkworm gut in the method of closure described would lead me to continue its use. At the same time it is well recognized that there are many other technics which have also proven quite satisfactory to their respective advocates.

Dr. J. W. Kennedy, of the Joseph Price Hospital in Philadelphia, reported in September, 1934, the combined experience of Dr. Price and himself, extending over a period of 56 years with over 30,000 laparotomies, without a single disruption. This remarkable record was obtained by the simple method of through and through interrupted silkworm gut suturing of abdominal wounds. No catgut was used and no buried silk, except in the large incisional ventral and umbilical hernias.

Dr. J. F. Baldwin, of Columbus, Ohio, reported in July, 1934, 35 years experience with 16,465 laparotomies without a single disruption. His wounds are closed with chromic catgut in layers reinforced by the use of silkworm gut tension sutures. Baldwin stresses the desirability of never removing the silkworm gut sutures before the fourteenth day, and leaves them longer if there is any suspicion of delayed healing. He further stresses the importance of the scultetus binder which is always firmly applied before our patients leave the operating room and the use of which is frequently continued for a week or two after discharge from the Hospital.

Conclusion

The evidence is ample to prove that there are certain constitutional factors which may retard the healing of a laparotomy wound. This has long been recognized and all efforts would be made to improve the patient's healing time by attention to these conditions.

However, it is believed that in spite of adverse constitutional factors, a laparotomy wound may be sutured with a technic which will eliminate the danger of disruption.

The technic here presented involves the use of a minimum amount of buried chromic catgut and the use of tension sutures of silkworm gut which are left in situ until the wound is healed.

With careful surgery and attention to details in the closure of laparotomy wounds, the disruption should become a surgical curiosity.

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THE DIAGNOSIS AND SURGICAL TREATMENT OF CARCINOMA OF THE LARYNX

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Baltimore

THE complete, early removal of cancer by surgical means still remains the most satisfactory method of treating this disease. In carcinoma of the larynx surgery is particularly successful, if employed early, because the growth can be easily removed. The type of carcinoma most frequently found in the larynx is the highly malignant squamous cell variety, which occurs in about 96 per cent of the cases. These patients usually die within one to three years unless the growth is removed.

About 4 per cent of all cases of cancer occur in the larynx. Ninety-eight per cent of the malignant tumors are carcinomas, 1 per cent adenocarcinomas, 1 per cent papillary carcinomas, and 2 per cent basal cell carcinomas.[1]

In the past few years so much has been written regarding the use of radium and x-ray in the treatment of cancer, that a great wave of enthusiasm spread over the country for more conservative ways of dealing with this disease. But time and experience have shown that radium and x-ray are not a cure-all for cancer, and that surgery still remains supreme.

The general use of x-ray before operation is inadvisable, as its action greatly complicates the removal of the growth and is often the cause of a delayed operation, which might otherwise have been successful if it had been performed in time.

X-ray does, however, have a definite place in treatment after the growth has been removed. It has a distinct value of preventing recurrence after operation.

DIAGNOSIS

Early diagnosis is by far the most important phase of this subject. A recognition of precancerous pathologic changes in the larynx by simple indirect laryngoscopic examination has saved many lives. A favorable prognosis depends much more upon a correct diagnosis in the early stages of the disease, before there is extension and metastasis, than upon the special type of operation selected or upon the unusual skill of an operator.

Diagnosis as a rule is not difficult to one who will give attention to well established symptoms, yet it is surprising how many of these cases are unrecognized until they have passed into advanced stages.

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

It is not uncommon for such patients to come in with extrinsic symptoms, giving a history of having been treated for months for hoarseness with ordinary sprays and local applications, while carcinoma was not suspected and no laryngeal examination was made.

One of the most important early symptoms of cancer of the larynx is some alteration in the voice of an adult. The disease is most common in males, and is usually seen in middle and later life.

Any person after the age of 40 who slowly or suddenly develops changes in his voice, such as weakness, huskiness, hoarseness or aphonia, without other special physical signs, should be suspected of having carcinoma. A careful laryngoscopic examination should be made and if there are suggestive changes in the larynx, every effort should be made to determine the true nature of the lesion.

The growth usually begins as a localized bulging on one of the vocal cords.[2] The surface of the cord is first attacked, but if the invasion continues the deeper tissues are involved with later extension to surrounding structures. Carcinoma of the larynx has a tendency to involve the anterior two-thirds of the cord, which is a helpful point in diagnosis. The local appearance of the lesion in incipient cancer of the larynx has several characteristic features.[1] It is seldom pedunculated, but may present a broken, irregular, warty appearance. The surrounding area is not inflamed, and may appear normal with little demarcation from the natural tissue. Its base may show vascularity and in later stages the growth may be "berry-like," uneven, infiltrated, with a bleeding surface. Rapid growths are often yellow or dirty white in color with a tendency to develop a cauliflower appearance.

In all cases of new growths in the larynx, the final decision must be made by taking a piece of the growth for microscopic study. This section is easily obtained through a direct laryngoscope, can be quickly made and is not dangerous to the patient.

There has been considerable controversy about the question of biopsy in these cases, but if the section is carefully made and the remaining raw edges of the growth are cauterized by endothermy, there is little danger that metastasis will develop. Of course, one should always be prepared to remove the growth as soon as possible after a definite diagnosis has been made.

There are several chronic laryngeal lesions which call for differential diagnosis. As laryngeal tuberculosis is one of the most common chronic laryngeal diseases, a few differential points of the two diseases will be considered.

Tuberculous laryngitis presents a typical picture.[3] It is a secondary manifestation and some pulmonary lesion is always associated. The

tubercle has no characteristic tendency to involve a certain part of the larynx as is seen in cancer. The lesion may be single or multiple, most often being seen in the arytenoids, the interarytenoid space, the vocal processes, one or both cords, the epiglottis, or in the pharynx. The early pathologic changes produce edema, infiltration and later breaking down with ulceration and secondary infection. The ulceration at first is superficial with later extension into the deeper structures, causing irregular loss of tissue. The vocal cords often present the typical "moth-eaten" appearance.

Chronic laryngitis in which there is some edema or infiltration of one of the vocal cords, in older persons, may at times present a serious problem in differential diagnosis.

Syphilis of the larynx has been uncommon in my practice. Hoarseness occurring in the secondary manifestations of the disease is generally associated with other general symptoms so that a diagnosis can be easily made. Furthermore, as this is a blood disease, there is usually uniform injection and infiltration of both cords. A Wassermann is also a great help. Cases causing most concern are those in which the patient has a true cancerous growth with syphilis. Many of these cases have been carried into the late stages of metastatic involvement by attempting to treat the lesion as syphilis. In all such cases the question of diagnosis should be decided by biopsy.

Treatment

The treatment of carcinoma of the larynx depends upon the individual patient, as no common method of procedure can be applied to every case. It is now generally recognized that there is considerable variation in the degrees of malignancy. The various lesions have been graded by Broders' method, so that a careful study of the type of cancer must be made before operation is decided upon.

Endolaryngeal operations have not been very successful in the treatment of cancer of the larynx and are now seldom attempted with the expectation of obtaining a cure. Likewise, treatment of such growths with radium has been disappointing, and it is now used as a palliative measure. No doubt the inaccessibility of the larynx for direct application has been the cause of failure in some cases. If large doses of radium could be applied directly to the growth as in skin cancer, the results would be better.

Laryngofissure is an ideal operation for early laryngeal cancer[4]—cases in which the growth is limited to the anterior surface or free edges of a mobile cord.[5] St. Clair Thomson states that 80 per cent of suitable cases can be cured by laryngofissure. This operation is safe, gives the patient a useful voice, free airway, and little disability, and should be used whenever possible. It is unfortunate that

only about 20 per cent of cases of laryngeal cancer are recognized early enough for this operation. The rest require a more radical procedure, such as laryngectomy.

Laryngectomy

In 1866, Watson of Edinburgh, was the first to attempt complete extirpation of the larynx in a patient suffering with syphilis.

The first total laryngectomy for cancer in man was performed by Billroth in 1873. From that time up to 1907, only 500 operations of this type had been reported. Early results were discouraging, complications common, and the mortality rate was high so that this operation made slow progress at first and was generally used as a last resort with little hope of curing the patient.

Today we know that laryngectomy can be safely performed by modern technic so that results compare favorably with those of any other surgical procedure, the mortality rate being less than 2 per cent.

Much credit must be given to Mackenty[6] for his contributions to this subject and to his perfection of a technic which was responsible for improvement in results. Since his time each surgeon has endeavored to add something to this operation to make it safer and more successful as to the final outcome. This has been my desire and I wish to record methods which have given me best results.

The single stage operation is usually employed. In most cases the cancerous growth can be removed at first operation. With some patients, where there is marked dyspnea, it is advisable to perform a preliminary tracheotomy, but these cases are in the minority.

Preoperative Attention.—As most cases of carcinoma of the larynx occur in males, late in life, much care must be given to the general condition of the patient. At this time of life complicating diseases are common. Cardiac lesions are frequent; nephritis is to be suspected, focal infections around the teeth, sinuses and tonsils should be eliminated, in fact every patient should be treated by an internist when necessary to get him in the best possible condition to stand a major operation. Good results cannot be expected if the patient is a poor operative risk and his condition should be improved before the operation is attempted.

Anesthesia

Before the perfection of rectal anesthesia, it was a common practice to perform most laryngectomy operations under local anesthesia. With gaseous anesthetics, usually employed, definite difficulties are added to the operation. The anesthetist is necessarily so close

to the field of action that he is in the way. Even extended rubber tubes embarrass the surgeon in his work. Most important of all, the concentrated fumes of ether on the mucosa of the lower trachea and bronchi in dehabilitated patients is always a matter of concern to most operators. These complications of course can be avoided by block and infiltration anesthesia, but local anesthesia also has its disadvantages. The patient is conscious, and this is a serious handicap. It is extremely depressing to most people to learn that they have cancer, but to know that they must lose their organ of voice is a real tragedy. So to lie on an operating table for several hours meditating on their misfortune, while their vocal cords are being removed, produces a mental shock and vasomotor disturbance which must affect the immediate recovery from the operation and certainly prolongs the convalescence.

By the use of rectal anesthesia many of these difficulties are overcome. I prefer avertin and invariably use it as a basal anesthetic.[7] In most cases the entire operation can be completed while the patient is under the influence of this drug alone. However, if necessary, a small quantity of ether in olive oil can be satisfactorily given per rectum as the particular case demands. With avertin the patient goes to sleep easily, quietly, and quickly. This is a very important factor and one which frequently prevents the necessity of a preliminary tracheotomy, which is required if there is excitement, struggling, or a spasm of the constricted glottis, a condition associated with the excitement stage of gaseous anesthetics. With avertin, the blood pressure is lowered, the breathing is quieter, and the patient sleeps for several hours, a very important advantage.

After using this method for several years, I am convinced that this is the ideal anesthetic for this operation and so far I have had no complications and have seen no bad effects from its use. There is one precaution, however, to be observed: the solution should never be given in the patient's room, but should always be administered on the operating table, because if dyspnea due to the growth should develop, an immediate tracheotomy can be performed more satisfactorily.

Operation

The skin (anterior part of neck and upper part of chest) is shaved and carefully scrubbed with soap and water. This can be done before the patient comes to the operating room if preferred. While the patient is going under the influence of avertin, the desired area is cleansed with alcohol, ether, and iodine, or Scott's solution, the application depending upon the choice of the operator. The neck is now extended as much as possible and the necessary draping arranged. Suction, with suitable small tubes, should be in

good working order. Naturally, a complete tracheotomy set should be at hand with the other required instruments.

Incision:—Particular attention should be paid to the preliminary skin incision.[8] I prefer a crescentic incision on the right side of the neck.[2] Beginning above, near the midline at the highest point of the right thyroid cartilage, it is extended downward and outward until the central part of the incision is on a line with the inner margin of the sternocleidomastoid muscle. From here the incision is carried downward and inward to the midline on a level with the first tracheal ring.

Such an incision, to my mind, has several advantages over the customary T-shaped incision. The most important feature of the crescentic incision is that there is less weakness in the midline of the throat, where strength and support is so essential after the larynx has been removed. Infection is less likely and healing is more rapid. On account of lessened traumatism and undisturbed blood supply over the esophageal region, fistulas do not develop so frequently. The larynx is easily exposed and satisfactorily delivered. There is less scarring with a final result of little deformity in the conspicuous midline of the throat.

The fascia and attached muscles are now gently retracted, giving a good exposure of the anterior part of the larynx. It is surprising how easy it is to deliver the larynx through this type incision, and it is unnecessary in most cases to employ wide, extensive, mutilating cutting, which for years has been so generally used. A vertical incision is now made in the midline and the surrounding dissection is kept as close as possible to the larynx proper. This close type of operation has several advantages. The healing is more rapid; there is little bleeding, which can easily be controlled; there is less tendency to pocket formation; the development of a fistula is rare; and the final cosmetic result is much better.

After the larynx is freed, it is rotated from side to side and the greater cornua of the thyroid cartilage are cut near the end. The superior thyroid artery and any other bleeding vessels are ligated.

The operation is performed in one stage unless there has been some unusual complication, which has required a previous tracheotomy. Continuing downward, the isthmus of the thyroid gland is now cut through its central part in a vertical plane. Through each cut end are inserted three chromic catgut, mattress sutures, the ends of the sutures being held by a clamp and retracted. Later on these edges are brought together and tied, forming a strong and added covering to the stump of the trachea.

The next step in the operation is of great importance, because much of the success of the operation depends upon its proper exe-

cution. The trachea is cut across from below upward at an oblique angle so as to leave an elongated posterior flap which is to be used later for a covering to the tracheal stump.

A pair of long, curved, sharp-pointed scissors is inserted through this cut end of the trachea, downward to a line with the third tracheal ring. At this point an opening is made through the trachea, connective tissue, and skin. This makes a new tracheotomy opening below with a bridge of attached tissue above to prevent infection and aids in better healing. The skin edges of this new tracheal opening are now carefully inverted and sutured with fine silk. After this a large tracheotomy tube is inserted and held in place by tape.

The upper end of the trachea now receives particular attention. The mucosa lining the interior is dissected upward and the edges carefully sutured in a vertical manner, completely closing the opening. Now the cartilaginous margins are closed over, with chromic catgut, from above downward in a horizontal plane, forming a second and secure covering to the entire upper part of the tracheal stump. The ends of the thyroid ligament are now brought together, and the mattress sutures previously inserted are tied forming an additional support to this area. This fills in, covers over, and protects a place in the operative field where infection was most likely to develop. By being closed off completely above, it prevents drainage into the trachea and bronchi and lessens the chances of lower respiratory tract infection, with pneumonia, the most common complication of this operation. Thereby being an important factor in improving the technic with greater chances for successful recovery.

The operation is now continued by lifting up the lower end of the larynx and carefully dissecting it posteriorly from below upward. Great care being used to prevent perforating the esophagus. After being freed from its attachments, the larynx is lifted out of its bed and a gauze handkerchief placed behind it. The upper end of the larynx is now severed and the entire mass is removed.

The success of every laryngectomy depends greatly upon the proper closure of the pharyngeal opening. This is the most vulnerable point of weakness, the most susceptible place for infection to develop with breaking down of tissue and fistula formation. In order to reinforce this particular area with an added layer of cartilage, in some cases, the epiglottis can be inverted, so that the tip is brought downward, the lower edges pared, and sutured into denuded esophageal margins. This makes a neat, easy closure and forms a mucosal lining to this important region. The outside, and remaining esophageal edges are now sutured vertically and horizontally in a T-shaped manner. Interrupted chromic catgut ligatures are used in this part of the operation. The remaining space

made by removal of the larynx is now closed over by carefully bringing the muscles and fascia together so as to form a strong wall of support. An effort is made to prevent pocket formation, so that a small vaseline drain is only required. This extends from the central part of the wound to its lower margin. The skin is now closed with fine silk sutures, reinforced by about three, deep, heavy silk sutures at points where there is likely to be tension.

The wound is covered with vaseline coated rubber tissue over which is placed a dressing of vaseline gauze. This to extend beneath and below the tracheotomy tube.

The tape on the tracheotomy tube is made taut and tied in a hard knot behind the neck.

It is extremely important to aspirate the trachea and bronchi with a soft rubber catheter attached to a suction apparatus, to remove blood clots or mucus which might be left in the bronchi.

The patient is usually given an intravenous solution of glucose before leaving the operating table.

After-Treatment in Laryngectomy

The after treatment of a laryngectomy patient is almost as important as the operation.[2]

Exacting vigilance is required with every case. The resident and nurse must be made to realize how much depends upon them for the patient's recovery. While returning from the operating room the patient's head should be lowered. A suction machine should be placed at the bedside for immediate action and should be used at all times when secretions accumulate in the trachea and bronchi. This exudate can be aspirated with a small curved cannula; this procedure will relieve the mucosa of infected material which is often the cause of one of the chief complications, broncho-pneumonia. Attention to this factor must be observed throughout the patient's convalescence. Dressings should be changed twice daily until the wound has healed. Iodine solution applied to the skin incision will prevent infection and stimulate healing.

In these cases proper feeding is essential. Concentrated liquids should be administered at frequent intervals through the feeding tube, to build up the required resistance. At the end of a week or ten days, the feeding tube can be removed and some sterile water can be taken by mouth as a test. If an esophageal fistula is present, the tube should be re-inserted until healing is complete.

Complications. Hemorrhage, usually considered the chief cause of anxiety, should not occur if the vessels have been carefully tied off at the time of the operation.

Bronchopneumonia can also be prevented by regular aspirations of secretions with a suction outfit. The patient should also be forced to change his position frequently and should be allowed to sit up in bed at intervals during the day.

Fistula formation, a common complication with the T-incision, is less likely to occur if there is strong support over the esophageal incision, employed with the technic described. If a fistula does develop, it is of no serious consequence and usually heals in a reasonable time, but if persistent, it can be closed under local anesthesia.

Summary

The early diagnosis of carcinoma of the larynx is most important, because if recognized early, the growth can be easily removed by a conservative operation. Hoarseness or any change in the voice of a patient over 40 years of age, without acute, or general symptoms, requires serious investigation, for by complete removal of laryngeal cancer many years can be added to the patient's life.

A diagnosis between laryngeal tuberculosis, chronic laryngitis, and syphilis of the larynx is easily differentiated. In all questionable cases a biopsy should be made.

Endolaryngeal operations have a very limited sphere of usefulness. Laryngofissure is an ideal operation for early cancer of the larynx but only about 20 per cent of the cases are obtained early enough for this type of operation. The remaining cases must be dealt with more radically, as about 80 per cent require laryngectomy.

Surgery still remains the most successful method of treating cancer. Radium cannot be successfully applied directly to laryngeal growths. Improvement in the surgical technic of laryngectomy has been perfected so that results from this operation compare favorably with that of any other surgical procedure. X-ray treatment of cancer of the larynx is of most value after the tumor has been removed and should not be used before operation.

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THE MASTOID AS A RESERVOIR IN CHRONIC OTORRHEA

Chronic Exudative Sclerosing Mastoiditis

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THE term "conservative" has been so widely used and so variably interpreted that it has become an unfortunate word in medical literature. The public and too many of the profession have considered the term "conservative" to mean *without surgery* and "radical" to mean *with surgery*. However, these classifications should be interpreted on the basis of what is to be conserved in the way of function and now how.

We have observed that often the residual hearing in the chronically diseased ear is given too little consideration in the planning of treatment. The preservation of hearing, even though impaired, is no less important than the immediate cessation of drainage and a closed drum. The importance of obtaining an intact drum cannot be too strongly emphasized. Larger perforations of the drum heal by marked scarring and distortion while the smaller ones heal almost without a trace. In both types, an effective barrier can be obtained and some of the function restored.

It is imperative to exercise the greatest care and thoroughness in studies of chronic disease of the ear. No regimen of treatment should be outlined that is not based upon a good physical examination, a careful laboratory study and above all x-ray of the mastoids. It is impossible to evaluate activity proximal to the middle ear without these studies. It is hoped that our paper will develop principles which can be accepted as indicating a proper line of treatment.

Certain chronic diseases of the ear respond well to simple intra-auricular medication and good hygiene. Many of those who do not will respond to conservative mastoidectomy. Certainly it is believed many of the radical mastoidectomies can be avoided in the specific type of disease under discussion. We shall be sorry if any indiscriminate surgeon or otologist uses this paper as an excuse for unnecessary otologic surgery.

SOURCE OF PAPER

This paper is based upon our private practice covering the period of 1930 to 1939. In this time we have performed 407 conservative

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and 48 radical mastoidectomies. Of this number only 17 fell into the classification of chronic exudative sclerosing mastoiditis. Of these 17 patients, 8 were males and 9 were females, whose ages ranged from 7 to 45 years, but predominantly they represent the first and second decades. The disease was unilateral in 14 and bilateral in 3 and involved the right ear in 11 cases and the left ear in 9.

History

The history is not particularly different from that obtained in the broad general classification of chronic diseases of the ear. However, we wish to discuss three symptoms, two of which are always present and one of which is only occasionally seen.

The discharge is mucopurulent but largely mucoid. It may be intermittent or constant. The odors are variable: odor is not an important sign in our opinion as to the character of the lesion; it more nearly represents the hygienic level of the patient and specifically the hygiene of his diseased ear.

Deafness is a most important factor. It is variable in the individual patients and changeable from time to time dependent upon the stage of disease, amount of secretions and apparently other physical factors not yet too well known, may be of influence.

Headache has been present in only two of our patients, one of whom had a peri-sinus abscess; we did not find an apparent reason for the second.

Infection of the upper respiratory tract apparently contributed to the onset of the otologic disease in 16 cases; one patient while diving had an accident to his ear. In none was there a background of an acute exanthema.

The method of myringotomy may on close analysis prove to be an important factor. In 17 cases under discussion 11 were spontaneous, 3 surgical, 1 was the result of a diving accident and 2 were of unknown origin. The preponderance of the spontaneous rupture of the membrane in this series may be significant.

The length of time otorrhea has been present does not seem to be particularly important. In this series the otorrhea ranged from 4 months to 20 years, averaging a little over 6 years. It is realized that an otorrhea of only four months might be classed as either acute or chronic. We have been more interested in presenting diseased changes than in the length of time the condition has existed and on this basis the otorrhea of four months was chronic.

Physical Examination

The canal is usually in good position but there is a variable amount of drainage and odor. The drum showed perforation in

every instance and there were four with polypoid tissue blocking the canal. Two perforations were central, two were posterior-superior in Shrapnell's membrane, six were along the floor central, two were posterior-inferior in pars flaccida, one in the superior portion of pars flaccida, one in the superior center of Shrapnell's membrane and one in the anterior-superior portion of Shrapnell's. Four were not interpreted because of blockage of the auricular canal by polyps. The literature leads one to believe that the perforation of the drum is an important index of what one can expect by certain therapy. This attitude we believe is due for a re-evaluation.

X-Ray Findings

It is quickly recognized that one cannot apply a rigid classification to any mastoid disease on x-ray. However we believe the following classification based upon surgical pathology can be used in the specific group we are presenting:

1. Exudative mastoiditis
2. Exudative sclerosing mastoiditis
3. Sclerosing mastoiditis.

In exudative mastoiditis, as the term indicates, there is a predominance of the exudative lesion over other changes interpretable by x-rays; the trabeculae are softened and in the cases under discussion several large lacunae were shown indicating a coalescence of proximal abscessed areas in the mastoid. The acini show a loss of aeration indicated by diffuse haziness.

The exudative sclerosing mastoid shows about an equal amount of exudate along with sclerosis, the trabeculae being in this instance hardened standing out sharply on the x-ray. The cell cavities show decreased illumination. The size of this mastoid has not been materially altered over its pre-infected size by the disease. This we consider an important point.

The sclerosing mastoid is the type that has destroyed all peripheral cells and has left only a small triangular area as outlined by the base of the middle fossa, the anterior wall of the lateral sinus and the posterior bony auricular canal. However in this sclerosed area, disease is still active and many small areas of exudate are found on operation. These "active" areas show as indistinct bone spaces with indefinite margins.

Surgery

It is sufficient to say that simple complete conservative mastoidectomy is the operation indicated in the type of disease under discussion. We would like to emphasize the importance of a posterior skin flap incision which avoids the hideous scar seen so often follow-

ing mastoidectomy. It has been our experience that a bad cosmetic result is almost as much a source of criticism by the patient as a bad clinical result. This the medical profession has not always sufficiently appreciated.

It will be necessary to do considerably more work along the antrotympanic bony septum than in the classical conservative mastoidectomy, thoroughly cleaning out the mastoid cells well ahead into the zygoma, along the floor of the middle fossa and proximal to the middle ear, or disease will persist. Not all conservative mastoidectomies are complete but they must be in these lesions.

Surgical Pathology

The surgical pathology as cited by various authors is not sufficiently specific in individual classifications to fit into the picture we are presenting.

Politzer[1] made some important observations that we closely relate to our problem. He stated that the most important changes of the mucous membrane in chronic diseases of the ear consist of general thickening which is due to an excessive infiltration of round cells and to the dilatation of blood vessels and the formation of new ones. Although the periosteal layer of the mucous membrane is pathologically altered, nevertheless it almost always remains intact and the subepithelial layer becomes so compressed through round cell infiltration that it is replaced by a suppurating granulation surface which is traversed by dilated, tortuous, partly newformed vessels. He also said that there is a dilatation of lymph vessels in the deeper vessels of the mucous membrane in chronic suppuration of the middle ear. They appear as varicose constricted vessels ending in clublike blind pouches and small branches which form an anastomosing network. He went on to discuss at length other changes which he thought lead to condensation of the osseous tissue, to hyperostoses and formation of osteophytes or to destruction or atrophy of the bone and finally to caries and necrosis of the temporal bone and ossicles with their unfortunate sequelae.

Jackson and Coates[2] also state that the mucous membrane is thickened and fibrous. The contours of the tympanic cavity are smoothed out and the niches are filled with hyperplastic tissue which shows a variable amount of small cell infiltration. Small cystic spaces are constantly found in which the vessels are dilated. In others the bone shows signs of caries. The small cell infiltration reaches down into the bone and into the marrow spaces and vascular channels. Osteoclasts are found in the dimples and hollows of the bone indicating active absorption.

In these two descriptions of pathology one can recognize the anatomic reasons for these changes.

Unfortunately we have not obtained any microscopic sections of this type of disease. At the time we were treating them we did not consider that we were dealing with a special classification that indicated a special study. We shall however obtain sections of this type for future study.

We have fairly accurate descriptions of the gross pathologic changes found at operation. The typical findings are those of transudate and exudate, this being limited to individual mastoid cells and to the coalescent areas which we have defined as lacunae. This exudate is variably described in our surgical notes as "gummy," "oleaginous" or "syrupy" which described after all a physical property easily understood. Is this unusually cohesive transudate or exudate secreted as such, or is it a result of absorption of serous contents?

The trabeculae as we have indicated may be sclerosed or softened.

The lateral sinus was uncovered by disease in two cases, one resulting in lateral sinus thrombosis and the other a low grade meningitis from a peri-sinus abscess; both patients recovered.

There were three who showed definite evidence of cholesteatoma.

Fourteen of the mastoids showed transudate and exudate with sclerosed trabeculae.

Five showed transudate and exudate with softened trabeculae.

Particular attention must be called to the fact that the two complications developed in mastoids which showed transudate and exudate with softened trabeculae. This suggests the importance of early recognition of a condition that must be viewed with grave alarm requiring accurate interpretation and selective treatment.

Pre-surgical Complications

The pre-surgical complications of mastoid disease and middle ear suppuration are too well known to permit of their inclusion in full in this article. However in the series under discussion there were two out of a total of seventeen, one developed a lateral sinus thrombosis and the other a peri-sinus abscess. However one must emphasize a most important pre-surgical complication, irremediable deafness. This has not been considered as a complication in otologic literature but in our opinion it deserves no other interpretation.

Results

Sixteen of these patients have obtained dry ears and healed drums: one has not due to poor choice of surgery. She should have

had a radical mastoidectomy at onset. There has been no further loss of hearing in any of the patients and thirteen have improved by approximately 20 per cent. There has been no re-infection of the ear or mastoid in the sixteen cases reported. No death occurred in the series. All of the sixteen patients are grateful for the effort and are satisfied with their results.

Case Reports

Case 1

Case 1.—Mrs. G. G., aged 28. Exudative lesion.

Duration of Otorrhea: 30 months.

X-Ray Interpretation: The left mastoid is well developed, nicely demonstrated and clear throughout. The right mastoid shows slight osteititis about the periphery posterior with a loss of cell definition throughout but with fairly good aeration posterior.

Impression: Exudative mastoiditis.

Treatment: Right, complete, conservative mastoidectomy.

Surgical Pathology: Immediately upon opening the mastoid cortex an oleaginous, amber-colored material under pressure literally herniated. The trabeculae were markedly softened and there were areas of lacunae throughout. One of these lacunae communicated with the lateral sinus and this lacuna was filled with black exudate resembling old blood.

Case 2.—Mrs. M. S., aged 44. A typical exudative type of lesion.

Duration of Otorrhea: 6 months.

X-Ray Interpretation: The left mastoid is extremely large, highly pneumatic and clear throughout. The right mastoid shows a restricted development or peripheral destruction. It is still well ventilated but there is loss of definition of the intersepta. The floor of the middle fossa and anterior wall of the lateral sinus are markedly exaggerated forming a faint triangle of sclerosing bone.

Treatment: Right, complete, conservative mastoidectomy; ligation of jugular vein; lateral sinus phlebotomy.

Surgical Pathology: Upon opening the mastoid cortex a thick, gummy, amber liquid escaped under pressure. The trabeculae had been markedly softened and in areas were absent. The lateral sinus was uncovered by disease for fully 2 cm.

CASE 3

CASE 3.—R. M., aged 20. Exudative type.

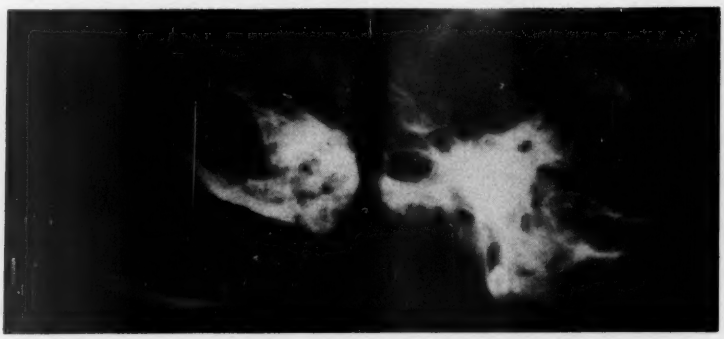
DURATION OF OTORRHEA: 4 months.

X-RAY INTERPRETATION: The left mastoid is highly pneumatic, well developed, nicely demonstrated and clear. The right mastoid shows diffuse opacity with cell trabeculae not visualized.

IMPRESSION: Subacute or chronic mastoiditis on the right of exudative character.

TREATMENT: Right, complete, conservative mastoidectomy.

SURGICAL PATHOLOGY: The mastoid cells had undergone softening of the trabeculae and the cell acini were filled with a syrupy, odorless, amber-colored fluid. Toward the antrum many of the cells had been broken down and this region was filled with granulation tissue which was slightly fibrous and bathed with mucopus. The mastoid was invaded throughout with these subacute or chronic changes.



CASE 4

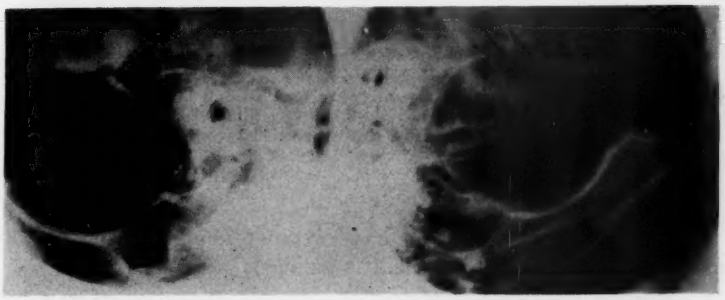
CASE 4.—Mrs. F. B., aged 23. Exudative sclerosing type.

DURATION OF OTORRHEA: 36 months.

X-RAY INTERPRETATION: The left mastoid is highly pneumatic, well developed and clear throughout. The right mastoid shows a restriction in its size with a loss of definition of the trabeculae and with considerable sclerosis of the floor of the middle fossa.

TREATMENT: Right, complete, conservative mastoidectomy.

SURGICAL PATHOLOGY: Upon opening the mastoid cortex, clear, thin, serous transudate escaped under pressure revealing thickened, sclerotic trabeculae of the various acini. The entire mastoid had undergone sclerosis of the trabeculae and the cell acini were flooded with the peculiar serous transudate described.



CASE 5

CASE 5.—Mrs. W. D., aged 38. Exudative sclerosing type.

DURATION OF OTORRHEA: 7 years.

X-RAY INTERPRETATION: The right mastoid is highly pneumatic, well developed and clear throughout. The left mastoid shows a few residual trabeculae which appear thickened and a few at their bases show wedge formation and evidence of an osteitis. Many of the cells have coalesced and there appears to be an exudate throughout.

TREATMENT: Left, complete, conservative mastoidectomy.

SURGICAL PATHOLOGY: Upon opening the mastoid cortex a serous, syrupy, gelatinous-like substance presented and this was found to have literally flooded the entire mastoid cell structures. The cells well ahead in the zygoma were filled with polypoid tissue. Proximal to the mastoid antrum these cell trabeculae exhibited sclerosis, being firm, friable and they fractured easily.

CASE 6

CASE 6.—Mrs. L. H., aged 25. Sclerotic type.

DURATION OF OTORRHEA: 20 years.

X-RAY INTERPRETATION: The left mastoid is well developed, nicely demonstrated and shows no frank alteration. The right mastoid shows definite sclerosis throughout with slight decreased density near the central posterior of the triangular area represented by the floor of middle fossa and anterior wall of the lateral sinus.

TREATMENT: Right, complete, conservative mastoidectomy.

SURGICAL PATHOLOGY: The entire mastoid process had undergone sclerosis and eburnation in all the mastoid except the region involving the antrum and proximal spaces for about 1.5 cm.; this was filled with a gelatinous, oleaginous, amber-colored fluid under pressure.

CASE 7.—C. K., aged 18. Typical sclerotic lesion.

DURATION OF OTORRHEA: 9 months.

X-RAY INTERPRETATION: The right mastoid is large, highly pneumatic and clear throughout. The left mastoid shows a triangular area-floor of middle fossa and anterior

wall of the lateral sinus which is apparently the confines of this mastoid. This triangular area shows some regions of aeration but no trabeculae are seen.

TREATMENT: Left, complete, conservative mastoidectomy.

SURGICAL PATHOLOGY: Upon opening the mastoid cortex, cancellous bone near the tip was encountered. This was followed upward and, about 1.5 cm. below the base of McEwen's triangle, cells were encountered which were filled with a transparent, unorganized, gummy, mucoid secretion. The trabeculae had become sclerosed, were quite hard and fractured stubbornly.

CONCLUSIONS

1. The chronic ear should have a most comprehensive survey before any regimen of treatment is seriously instituted.

2. Some mastoids do act as a reservoir in chronic otorrhea and many of these respond to conservative mastoidectomy.

3. The drum perforation is variable in position and size in this type of lesion.

4. The hearing is improved markedly following mastoidectomy in the presence of this specific classification of aural disease.

5. The drum-membrane heals following complete conservative mastoidectomy in this specific condition.

6. The chronic otorrhea in the presence of a "reservoir mastoid" apparently remains "quiet" if not actually healed following a complete conservative mastoidectomy.

7. The chronic ear in the presence of a reservoir mastoid is an active source of dangerous complications to adjacent structures.

8. The spontaneous ruptured drum predominates in this series, suggesting possibly an important factor in mastoid invasion.

9. No new idea nor originality is claimed but it is felt that by bringing this specific type of aural disease into sharp focus again renewed effective interest may be stimulated.

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SULFANILAMIDE AND STAPHYLOCOCCUS ANTITOXIN

Report of their Combined Use in the Successful Treatment of Staphylococcus Septicemia

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and

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A TEXTILE worker, aged 35, twisted his right knee painfully while at work on Jan. 10, 1939. He was able to walk home but during the night the knee become swollen and painful.

When seen the following morning, he was in bed complaining of pain in the right knee. Although this was tender and swollen there was no evidence of external trauma and there was no redness or swelling of the leg or foot. There was however a crusted ulcer on his head which he stated had been draining pus for some time. Also there were blisters on each heel, but they were not draining, though the skin was broken on the left heel.

On January 12, x-rays of the knee were negative. He complained a great deal while in the office and stated that the pain was growing more severe. The next morning the family reported that he had rested little during the night and seemed irrational at times. Examination showed increased swelling of the knee but no fluctuation. His temperature was 103 and the patient was unable to cooperate or answer questions well.

On January 14 the patient was delirious and moderately cyanotic. The temperature was 103. The right calf and ankle were slightly red and swollen. There were several petechiae on his hands.

A blood culture was taken at this time and the patient was started on 40 grains of sulfanilamide at four hour intervals. The blood culture was positive for staphylococcus and a second culture a day later was also positive.

On January 15, he was delirious and semi-comatose. At this time the knee was fluctuant and pus was obtained on aspiration which revealed a staphylococcus similar to the one found in the blood on two examinations. The knee was x-rayed again but no bone involvement was found. The knee was drained of about 3 ounces of pus and the incision was packed lightly and left open.

Blood culture taken the evening of the 16th was negative. Twenty-four hours later staphylococcus antitoxin was started. This and the sulfanilamide were continued and by the 20th the patient had received 180,000 units of staphylococcus antitoxin and 740 grains of sulfanilamide. On January 18, the temperature began to fall and on the 21st he became clearer and more cooperative.

During this time the knee drained freely. Leukocyte counts during the course of illness ranged from 20,000 to 40,000 and the sulfanilamide blood concentration ranged from 12 to 18 mg. per 100 c.c. of blood.

The patient continued to improve and by January 23 there had been three negative blood cultures. On February 7 he was sent home. On January 26 he was started on small doses of autogenous vaccine and by February 24 he was receiving 1 c.c. every third day. The patient was last seen on April 3,

1940 and was in excellent health with no symptoms or signs of a recurrence of the infection. There was some disability of the right knee but otherwise he was in perfect health.

Comment

Three things should be stressed in the treatment of staphylococcus septicemias:

1. Thorough drainage of primary and secondary foci as soon as localization has taken place;

2. The action of sulfanilamide on staphylococcus. Blood culture three days after beginning sulfanilamide was negative whereas two previous cultures had been positive. The first negative culture was obtained before administering the staphylococcus antitoxin;

3. The maintenance of a high concentration of sulfanilamide in the blood at all times.

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WATER BALANCE

Since the work of Cannon, Blalock and others on the mechanism and treatment of shock there has been an increasing tendency on the part of most surgeons to administer large amounts of fluids subcutaneously, by vein and by proctoclysis as a routine postoperative measure. There can be no doubt that these treatments have saved many lives and have reduced materially the operative mortality rate. But the very fact of the usefulness of fluid administration has led to overenthusiastic and sometimes indiscriminate usage.

Recent studies by many investigators, notably Newburgh and Orr, have demonstrated the necessity for a better understanding of the body water balance. They were able to show that profound changes in the chemical equilibrium of tissues occurred through hyperhydration as well as by hypohydration.

It is now well known that a body fluid loss equal to 6 per cent of the body weight produces clinical dehydration; a fluid loss of 10 per cent of body weight is fatal. On the other hand the waterlogged patient is not infrequently seen. There have been several cases reported in the literature of water intoxication. This condition, usually produced by the administration of large amounts of tap water orally or by proctoclysis, is a pathologic entity and unless the proper treatment is instituted is usually fatal. It is characterized by symptoms not unlike the uremic syndrome, namely, increasing stupor, disorientation and anasarca. In a large measure it is due to marked depletion of the blood chlorides and a concomitant acidosis. Determination of blood chlorides and the CO_2 combining power and the presence of acetone in urine will usually lead to the correct diagnosis. Treatment of course is aimed at reestablishing the nor-

mal chloride level and maintaining the acid base balance. Injection of hypertonic salt solution will frequently give a dramatic response. Fortunately most physicians employ physiologic saline for fluid injections and water intoxication practically never occurs when this is given. An excess of glucose or saline will by immobilization of tissue water result in edema, however. A damaged heart may be thrown into the initial attack of congestive failure by overloading the circulation. Similarly a renal insufficiency may be precipitated through edema of the kidneys.

A great many facts have recently been brought to light concerning the injection or ingestion of glucose as a solitary feeding agent. A previously healthy individual may be thrown into acute avitaminosis through four or five days of glucose feeding. This is accomplished by the loss of the coenzymic action of the B group vitamins which play such an important role in the carbohydrate breakdown and synthesis. It is now felt that a large percentage of the so-called cases of toxic psychosis which are characterized by stupor, clouding of consciousness, grasping reflexes and delirium are manifestations of nicotinic acid deficiency. Not infrequently acute avitaminosis will pass unrecognized or be confused with uremia or cerebral accident. Most of these patients will reveal the glossitis typical of riboflavin or nicotinic acid deficiency and once the possibility of a deficiency is kept in mind, treatment can many times prevent a fatal outcome.

Heyd in a recent paper which appeared in THE SOUTHERN SURGEON emphasized the usefulness of charting of the fluid intake and output. By keeping a careful record of the fluid intake and measurable output, and estimating the insensible loss that occurs through respiration, perspiration and feces, a fairly accurate method of estimating fluid requirements is obtained. An easy rule to follow is if the urinary output is below 500 c.c. per twenty-four hours, a state of dehydration and probably nitrogen retention exists. This is the approximate amount of urine which, with a specific gravity of 1.030, is necessary to remove the solid waste matter.

The use of fluids is a most valuable aid in treatment, but in a familiar everyday procedure we are likely to forget the ill effects which may be produced by improper administration. That fluids are beneficial, and many times life saving, everyone knows, but it appears that only a few are familiar with the hazards of uncontrolled dosage. The rational usage does not require a complicated set-up but only a competent nursing force who will find it a minor task to chart the intake and output. A knowledge of the fundamentals of water balance is all the physician needs in order to avoid these hazards.

BERNARD P. WOLFF, M. D.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

OPERATIVE SURGERY. By J. SHELTON HORSLEY, M. D., LL. D., F. A. C. S., Attending Surgeon, St. Elizabeth's Hospital, Richmond; and ISAAC A. BIGGER, M. D., Professor of Surgery, Medical College of Virginia, Surgeon-in-Chief, Medical College of Virginia Hospitals, Richmond. With Contributions by C. C. COLEMAN, M. D., F. A. C. S.; JOHN S. HORSLEY, JR., M. D.; AUSTIN I. DODSON, M. D., F. A. C. S.; and DONALD M. FAULKNER, M. D. In Two Volumes. Fifth Edition, Revised and Enlarged. 1650 pages, with 1391 illustrations. Price, $18. St. Louis: The C. V. Mosby Company, 1940.

These volumes improve with each new edition. It is doubtful if a better work on operative surgery has ever been published. One of the main attractions to greet the reviewer on first opening the books is the freshness of all the material, a vast improvement over textbooks of other days on the same subject. Older volumes were wont to devote too much space to obsolete methods. After giving a few lines on symptoms and indications for operation Horsley and Bigger at once describe the latest and presumably the best technic for surgical attack upon most of the diseases of human flesh generally considered amenable to such treatment.

The publisher is to be commended for continuing his presentation of new editions at such frequent intervals. This indicates the lively demand for the books and demonstrates Mosby's desire to keep up to the minute. At the same time strenuous responsibility is placed upon the various authors who manifestly can never get Operative Surgery off their minds. They must forever be getting ready to publish a new edition. For this thousands of students and readers should be profoundly grateful.

It strikes us that all of Mosby's books are better put together today than in former times. The present volumes leave nothing to be desired in this respect, while the large type is grateful to presbyopic eyes. In our review of the Fourth Edition considerable space was given to praise of Miss Lorraine's illustrations. The same praise applies to the new books. No textbook requires good drawings as much as a work on operative surgery, and these volumes are perfection in this regard. Original drawings also are essential, and Miss Lorraine has added 183 new illustrations to the Fifth Edition.

With a surgeon devoting as much time to the thorax as Dr. Bigger, a large amount of new material on operations on the chest should be expected and it is found in the new books. Nothing seems to be omitted. The rewritten chapters on arterial surgery are especially interesting and informative. In the section on neurology a full description is given of the lesions of the intervertebral discs and hypertrophy of the ligamentum flavum with the methods of diagnosis and the operations for this condition. The division of the sympathetic and parasympathetic nerves in the root of the lung for bronchial asthma is described.

Among new procedures included are ligation of the patent ductus arteriosus, segmental pneumonectomy and extrapleural pneumothorax. In the field of intravenous therapy are described the methods of blood banking, the use of Mueller's solution which contains alcohol, and Elman and Weiner's method of giving amino-acids for protein replacement. Much new material

has been introduced in the section on orthopedics, while the pages devoted to plastic surgery cover the subject completely.

While the excellent chapters of Dr. C. C. Coleman in neurosurgery, John S. Horsley, Jr., in plastic surgery, Austin I. Dodson in urology, and Donald M. Faulkner in orthopedics occupy increasing space with each succeeding edition, the contributions of the "old master," and originator of the book, Dr. J. S. Horsley, Sr., yet are outstanding and paramount, as should be expected. After treating of surgical principles, in which he always has been a leading authority, and discussing cancer and blood vessel surgery, his other pages are turned over to the ever important matter of abdominal surgery. The chapter dealing with peritonitis has been rewritten. In all the realm of surgical literature one cannot find clearer accounts of the operative procedures to be practiced upon the stomach and other parts of the digestive system than those furnished by Dr. Horsley in this Fifth Edition of the Operative Surgery.

Altogether this book is one for every medical library, private and public. While it serves primarily the needs of the operating surgeon, men in other lines often will profit by referring to its pages when studying the indications for surgical intervention and the results to be expected. The various subjects are treated with just the right amount of detail so that descriptions are quickly read and understood. After we all have had the opportunity properly to assimilate the fifteen hundred delightful pages of these two volumes, and their makers have had time to catch their breath, we shall be ready to review the Sixth Edition.

FRANK K. BOLAND, M. D.

DIAGNOSIS AND TREATMENT OF HEAD INJURIES. By SIDNEY D. GROSS, M. D., F. A. C. S., Attending Neurosurgeon, Beth Israel Hospital; Associate Neurosurgeon, Morrisiania City Hospital; Adjunct Neurosurgeon, Mount Sinai Hospital; Instructor, New York University, New York, and WILLIAM EHRLICH, M. D., Associate Attending Neurosurgeon, Newark Beth Israel Hospital; Neurosurgeon, Barnett Memorial Hospital, Paterson; Consulting Neurosurgeon, St. Joseph's Hospital, Paterson, New Jersey. Introduction by PERCIVAL BAILEY, M. D., Ph. D., Professor of Neurology and Neurosurgery, University of Illinois, Chicago. 275 pages, with 94 illustrations. Price, $5. New York and London: Paul B. Hoeber, Inc., 1940.

As Percival Bailey (who has given us a paper for early publication) points out in his introduction to this work, the modern automobile has made head injuries of concern to almost everyone practicing medicine nowadays. "No longer are seriously injured patients kept waiting for x-ray photographs of the skull to be made; these are made only for definite reasons, after all symptoms of shock have disappeared, often just before discharge simply because they may be needed as evidence in court." The present emphasis is on recognizing the nature of the injury, especially of the damage to the brain and treating the patient properly. "The usual subtemporal decompression is futile and only increases the damage."

In this book is set forth clearly with many illustrations what the general surgeon should know about head injuries. Its widespread study should serve to save many lives. We agree with the medical editor of TIME that it is most important and one that we recommend with enthusiasm to our readers.

A TEXTBOOK OF SURGERY. By JOHN HOMANS, M. D., Clinical Professor of Surgery. Compiled from Lectures and Other Writings of Various Members of The Surgical Department of The Harvard Medical School. With a Special Bibliographical Index and With Illustrations by WILLARD C. SHEPHERD. Fifth Edition, Completely Revised. 1,272 pages, with 530 illustrations. Price, $8. Springfield and Baltimore: Charles C Thomas, Publisher, 1940.

Though Francis Bacon—or was it Aristotle?—took all knowledge for his province, in this late day no human mind can be capable of writing authoritatively of all surgery. To have various men deal with different subjects tends to unevenness of excellence and thoroughness, not to speak of repetition. Dr. Homans, it seems to us, has hit on the happy compromise of securing material from a whole surgical faculty and rewriting it in his own most pleasing style, thus securing a rare balance.

Several earlier editions of this textbook of surgery have exhausted the superlatives at our command: it remains an ideal text for undergraduates, and general practitioners can also learn from it what they need to know of surgery. The fifth edition contains the equivalent of more than 50 pages of new material concerning about 150 subjects, and yet the author has dropped enough old stuff to maintain the original pagination. A number of publications in 1939 have been reviewed and it would appear that the work has been brought thoroughly up to date.

We must issue a word of warning however: there are so many interesting bits of history, so many unexpected spots of entertainment that this book is hard to put down, so don't leave it on your bedside table if you have a lot of work to do the next day.

MINOR SURGERY. By FREDERICK CHRISTOPHER, S. B., M. D., F. A. C. S., Associate Professor of Surgery at the Northwestern University Medical School, Chicago, Chief Surgeon at the Evanston (Ill.) Hospital. Fourth Edition. 946 pages, with 639 illustrations. Price, $10. Philadelphia and London: W. B. Saunders Company, 1940.

Dr. Christopher has long been a leader in the field of minor surgery. His textbook is well known as a complete and reliable authority. The 1940 edition has been thoroughly revised, old material has been discarded and much new material added, so that it fully meets the high standards set by previous editions.

Fortunately, most of those who undertake major surgery have had a thorough training in surgical principles and technic. The same cannot be said of many of those who perform minor surgery. The result, as Dr. Kanavel has so well expressed it, is that "too often minor surgery becomes major surgery through carelessness or incompetence."

The borders of the field of minor surgery are indistinct and it is often difficult to determine what materials should be included in such a text. Dr. Christopher has chosen well his subject matter. That the fields of urology, gynecology, and proctology should be encroached upon is necessary for the sake of thoroughness.

The chapter dealing with open wounds fills a long felt need. Dr. Christopher's treatment in which he advocates the thorough cleansing of fresh

wounds with soap and water and the avoidance of harmful antiseptics cannot be too highly praised. It is a lamentable fact that the first thought of practically every layman and unfortunately many doctors is to swab the wound with any and every kind of antiseptic, preferably one with a brilliant color.

The approximation of wound edges by adhesive strips instead of sutures in certain selected cases merits greater attention than it has received in the past. The chapters dealing with circulatory disturbances, and the anal canal and rectum are excellent.

Lengthy consideration is given to injuries, infection, tumors, and deformities of the extremities. Fractures are more thoroughly discussed than is usual in a book of this type. The last chapter devoted to The Surgical Intern should be read and digested by every medical student before he enters his internship.

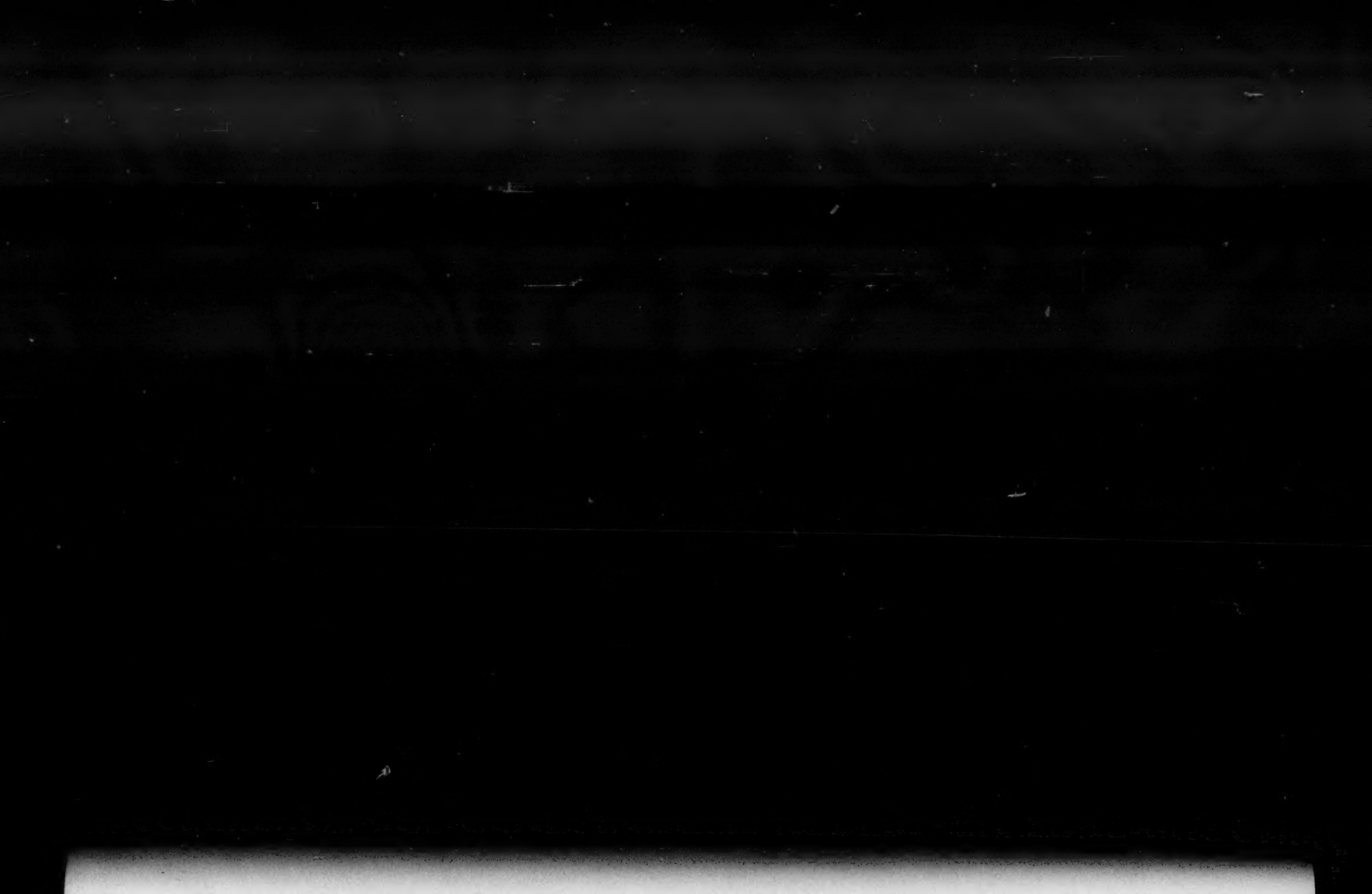
The illustrations are numerous, clear cut and well chosen. The type is easy on the eye. A voluminous bibliography is readily available.

This reviewer regrets that such an excellent book seems to advocate the treatment of frost bite by the antiquated and dangerous method of rubbing the affected part with snow.

All who do any type of minor surgery can greatly benefit by the use of this book.

GEORGE F. ARCHER, M. D.

A TEXTBOOK OF PATHOLOGY. By W. G. MACCALLUM, Professor of Pathology and Bacteriology, The Johns Hopkins University, Baltimore. Seventh Edition, Thoroughly Revised. 1302 pages, with 697 illustrations. Price, $10. Philadelphia and London: W. B. Saunders Company, 1940.

It is a pleasure to pick up a new edition of MacCallum for a score of years ago it was our favorite textbook of pathology. While a few new illustrations have been added, recognizing the original ones is like greeting old friends. We would not intimate that the age of the illustrations is a defect in the book; to the contrary these objective portrayals probably cannot be improved on—at least in our opinion they have not yet been equaled. The text however has been very extensively revised: some new sections have been added and it is surprising to see how many references there are to the literature of the world since the last edition. In passing, it is pleasant to be reminded that some Germans in the past few years have been able to carry on the traditions of medical research that characterized the glorious Germany of thirty years ago.

In short this classic is still at the top of the heap.